A2623-1

Public Health

EDITORIALS

What Infant Mortality Tells Us 653
Alfred Yankauer

Jails and Prisons: The New Asylums? 655
Douglas Shenson, Nancy Dubler, and David Michaels

Using Nature to Understand Nurture 657
Theodore B. Vanitallie and Albert J. Stunkard

COMMENTARY

The Opium Wars Revisited as US Forces Tobacco Exports in Asia 659
Ted T. L. Chen and Alvin E. Winder

ARTICLES

The Prevalence of Severe Mental Disorder among Male Urban Jail Detainees 663
Linda A. Teplin

Case-Control Study of Lung Cancer and Truck Driving in the Teamsters Union 670
N. Kyle Steenland, Debra T. Silverman, and Richard W. Hornung

Nongenetic Influences of Obesity on CVD Risk Factors: Analysis of Identical Twins 677
Beth Newman, Joseph V. Selby, Charles P. Quesenberry, Mary-Claire King, Gary D. Friedman, and Richard R. Fabsitz

Effect of Traditional Risk Factors on Infant Birthweight among Blacks and Whites in Chicago. 679
James W. Collins, Jr. and Richard J. David

The Dramatic Increase in the Rate of LBW in New York City 682
Theodore Joyce

Secular Change in Birthweight among Southeast Asian Immigrants to the United States 685
De-Kun Li, Hanyu Ni, Stephen M. Schwartz, and Janet R. Daling

Potential Exposure and Health Risks of Infants from Indoor Residential Pesticide Applications 689
Richard A. Fenske, Kathleen G. Black, Kenneth P. Elkner, Chorng-Il Lee, Mark M. Methner, and Ralph Soto

Breastfeeding Patterns in Puerto Rico 694
Jose E. Becerra and Jack C. Smith

Dietary Intake of Americans Reporting Adherence to a Low Cholesterol Diet (NHANES II) 698
Gordon Schectman, W. Paul McKinney, Joan Pleuss, and Raymond G. Hoffman

Spanish Version of the Nottingham Health Profile: Translation and Preliminary Validity 704
Jordi Alonso, Josep Maria Anto, and Conchi Moreno

PUBLIC HEALTH BRIEFS

Evaluating Community-based Nutrition Programs: Assessing Grocery Store Product Displays 709
Allen Cheadle, Bruce Psaty, Edward Wagner, Paula Diehr, Thomas Koepsell, Susan Curry, and Michael Von Korff

Poison Control Center Follow-up of Occupational Disease 711
Eddy A. Bresnitz

Survey of State Regulation of Testing for Drugs of Abuse Outside of Licensed Clinical Laboratories. 713
Daniel M. Baer, Richard E. Belsey, and Michael R. Skeels

Illegal Abortion in Mexico: Client Perceptions 715
Susan Pick de Weiss and Henry P. David

Survey of State Medicaid Coverage of Abortion and Prenatal Diagnostic Procedures 717
Janet Weiner and Barbara A. Bernhardt

Obstetrical Pulmonary Embolism Mortality, United States, 1970–85 720
Adele L. Franks, Hani K. Atrash, Herschel W. Lawson, and Karen S. Colberg

(continued page 643)

Featuring. . .

Maternal & Child Health

ED KIANKA
CONGRESSIONAL INFORMATION SVC 0086389 028
4520 EAST-WEST HIGHWAY
BETHESDA MD 20814

ociation 118th Annual Meeting
4, 1990 • New York City

SERVING SOCIETY–
PROTECTING HEALTH

AMERICAN JOURNAL OF Public Health

June 1990

Contents

Volume 80, Number 6

(continued)

Cervix and Breast Cancer Incidence in Immigrant Caribbean Women 722
Rachel G. Fruchter, Kamran Nayeri, Jean Claude Remy, Carolyn Wright, Joseph G. Feldman, John G. Boyce, and William S. Burnett

Ten-Year Changes in Birthweight Distributions of Black and White Infants, South Carolina 724
Robert M. Mayberry and Robert F. Lewis

Unlicensed Midwifery Practice in Washington State 726
Susan J. Myers, Patricia A. St. Clair, Stephen S. Gloyd, Philip Salzberg, and Joanne Myers-Ciecko

Occupational Physical Activity and other Risk Factors for Preterm Birth among US Army Primigravidas 728
Gilbert Ramirez, Richard M. Grimes, John F. Annegers, Barry R. Davis, and Carl H. Slater

Pawtucket Heart Health Program Point-of-Purchase Nutrition Education Program in Supermarkets 730
Mary K. Hunt, Craig Lefebvre, Mary Lynne Hixson, Stephen W. Banspach, Annlouise R. Assaf, and Richard A. Carleton

Infant Feeding Practices: An Evaluation of the Impact of a Health Education Course 732
Ilana Shoham-Yakubovich, Joseph S. Pliskin, and Daniel Carr

Pertussis Immunization in Eight-Month-Old Children in North Carolina 734
Dennis A. Clements, Catherine M. Wilfert, J. Newton MacCormack, Kristen A. Weigle, and Floyd W. Denny

Loss of Maternally Acquired Measles Antibodies in Well-Nourished Infants; Response to Measles Vaccination, Peru 736
Abraham Vaisberg, Jose O. Alvarez, Herminio Hernandez, Daniel Guillen, Pablo Chu, and Ana Colarossi

The Prime Time Diet: Eating Behavior and Food Messages in Television Program Content and Commercials 738
Mary Story and Patricia Faulkner

Seat Belt Use on Interstate Highways 740
JoAnn K. Wells, Allan F. Williams, and Adrian K. Lund

NOTES FROM THE FIELD

Diapering Decisions: A Community Education Project 743
Janet Primomo

The South Florida Perinatal Network 744
Marilyn Lesser and Patricia McGovern

LETTERS TO THE EDITOR

Quality of Death Certificates: Studying or Burying? 751
L. Rachid Salmi, Francois Dabis, and Tom McKinley
Response from Dr. Moriyama
Comments from NCHS/Rosenberg 752
Methods Omitted to Calculate Confidence Intervals 752
Kevin M. Sullivan
Response from Dr. Murray

ERRATUM

In: Evans, MR, *et al:* Potential for laboratory exposures to biohazardous agents found in the blood; April 1990 AJPH 658

JOURNAL DEPARTMENTS

Advertisers' Index 644
Masthead 644
Annual Meeting Forms, Late Breaker Sessions 693, 753, 755
Job Opportunities 759

The American Journal of Public Health,
published monthly,
is the official Journal
of the American Public Health Association,
1015 Fifteenth Street, NW,
Washington, DC 20005.
(202) 789-5600
APHA is a nongovernmental
professional society
founded in 1872,
representing all disciplines
and specialties in public health.

APHA OFFICERS
Myron Allukian, Jr., DDS, MPH, *President*
Iris R. Shannon, RN, PhD, *Immediate Past President*
C. William Keck, MD, MPH, *President-Elect*
Henrik Blum, MD, MPH, *Vice-President (USA)*
Betty Havens, MA, *Vice-President (Canada)*
Herman Vargas-Martinez, MD, *Vice-President (Latin America)*
Caswell A. Evans, Jr., DDS, MPH, *Treasurer*
Shirley Randolph, MSPH, *Chair, Executive Board*
George E. Hardy, Jr., MD, MPH, *Speaker, Governing Council*
William H. McBeath, MD, MPH, *Executive Director*

EXECUTIVE BOARD
Shirley Randolph, MSPH, (1991) *Chairperson*
Marilyn Aguirre-Molina, MS, EdD, (1993)
Myron Allukian, Jr., DDS, MPH, *President*
Ted L. Chen, PhD, MPH, (1990)
Molly Joel Coye, MD, MPH, (1991)
Brian A. Dobrow, MPH, (1992)
Caswell A. Evans, Jr., DDS, MPH, *Treasurer*
Eugene Feingold, PhD, (1991)
Janet Heinrich, MPH, DrPH, (1993)
Margaret H. Jordan, RN, MPH, (1992)
C. William Keck, MD, MPH, *President-Elect*
Allen N. Koplin, MD, MPH, (1990)
Joyce C. Lashof, MD, (1990)
Barry S. Levy, MD, MPH, (1993)
Iris R. Shannon, RN, PhD, *Immediate Past President*
Frances M. Veverka, MPH, RS, (1992)

EX OFFICIO
William H. McBeath, MD, MPH, *Executive Director*
Martha Katz, *Chair, Action Board*
George Pickett, MD, MPH, *Chair, Program Development Board*
George E. Hardy, Jr., MD, MPH, *Speaker, Governing Council*

37

The Prevalence of Severe Mental Disorder Among Male Urban Jail Detainees: Comparison with the Epidemiologic Catchment Area Program

LINDA A. TEPLIN, PHD

Abstract: This paper presents the prevalence rates of schizophrenia and major affective disorders by age and race among a random sample of male jail detainees. Subjects were administered the National Institute of Mental Health Diagnostic Interview Schedule (NIMH-DIS). The jail prevalence rates were then compared with general population data from the five-city Epidemiologic Catchment Area program using difference of proportion tests and loglinear analysis. After controlling for demographic differences between the jail and five-city samples, the jail prevalence rates were still two to three times higher than those in the general population. These findings suggest several public policy modifications concerning the psychiatric management of our burgeoning jail population. (*Am J Public Health* 1990; 80:663–669.)

Introduction

Mental health professionals speculate that the jails have become a repository for the severely mentally ill. Often referred to as the criminalization hypothesis, this trend is thought to be the unintended consequence of policy modifications, e.g., deinstitutionalization and more stringent commitment criteria.[1–3] Of particular concern are minor offenders (e.g., shoplifters) who could be treated in the mental health system but are instead managed by the criminal justice system. Criminal processing of the mentally ill may be most common among the individuals of the underclass because they have less access to treatment, fewer treatment alternatives, and less social support than wealthier persons.[4]

Jails, rather than prisons, are the critical point to gather psychiatric epidemiological data. Jail populations include detainees awaiting trial and convicted offenders serving sentences of less than one year, while prisons contain only convicted criminals serving longer sentences. Prison samples are biased because inmates are often diverted to forensic psychiatric facilities prior to conviction or imprisonment; prevalence rates of severe disorders in prisons[*] appear to be lower than those in jails[2] and those in the general population.[5,6]

The Appendix displays the findings of previous prevalence studies of mental illness among jail populations.[7–24] Interestingly, the studies listed show no increase in prevalence rates over time, and little consistency across studies. The tremendous variation in prevalence rates may be explained, in part, by three major methodological limitations:

- *Sample Selection*—To date, only four studies have used random samples.[11–13,23] One study used volunteers,[20] resulting in a plethora of potential biases. The most common type of study includes only persons who are referred for a mental health evaluation, and is obviously biased.
- *Measurement*—The variability in prevalence rates may be a result of the unknown reliability of the instrumentation used in many investigations. Since many studies used unspecified diagnostic criteria and/or nonstandardized instruments,[11,14,16] the diagnostic process is probably inconsistent across samples.
- *Sample Size*—Psychotic disorders have an extremely low base rate, between 1–2 percent.[5,6] The low base rate requires a relatively large sample size to generate accurate prevalence statistics.[25] No study incorporating a random sampling strategy used a large enough sample size.

None of these studies compare the jail prevalence rates with those in the general population. Moreover, because jail detainees are disproportionately young and minority group members[26]—characteristics correlated with severe mental disorder[27]—any comparison with baseline data must control for the jail's demographic composition.

The dearth of reliable psychiatric epidemiological data is critical because of the correctional system's overwhelming growth: between 1978 and 1983, the jail census rose by 40 percent, and by 1987, it increased by another 28 percent.[28–30] Research and intervention at the jail level are particularly important because not only are many jail detainees in need of ameliorative treatment, they are captive and physically amenable to intervention.

This paper provides an accurate estimate of severe mental disorder among male jail detainees in Cook County, Illinois, and compares the prevalence rate to that of the general population.

Methods

Subjects

Jail subjects studied were a stratified random sample (approximately one-half misdemeanants, one-half felons) of male detainees who entered Cook County Department of Corrections in Chicago, Illinois, directly from pretrial arraignment between November 1983, and November 1984 (N = 728). Persons charged with both misdemeanors and felonies were categorized as felons. Data were subsequently weighted to reflect the actual composition of misdemeanants and felons. The jail receives approximately 60,000 admissions per year[31] and is demographically similar to other large urban jails on such variables as race and age.[32] The size of the facility ensured adequate subject availability.

By assuming a binomial distribution for the frequencies of occurrence or non-occurrence, this sample size allows us to reliably detect events which have a base rate in the general population of less than 1 percent.[33]

Address reprint requests to Linda A. Teplin, Associate Professor, Northwestern University Medical School, and Coordinator, Psycho-legal Studies Program, Northwestern Memorial Hospital, 215 East Chicago Avenue, Room 708, Chicago, IL 60611. This paper, submitted to the Journal April 11, 1989, was revised and accepted for publication December 27, 1989.

Editor's Note: See also related editorial p 655 this issue.

[*] Collins JJ, Schlenger WE: The prevalence of psychiatric disorder among admissions to prison. Paper presented at annual meeting of the American Society of Criminology, Denver, CO, November 1983.

The age range of subjects was 16–68 years, mean and median age of 26.3 and 25.0 years, respectively; 57.4 percent were employed at the time of arrest. The racial composition was predominantly Black (80.8 percent); 6.5 percent of the sample were Hispanic, 12 percent were other Whites, and the remaining 0.8 percent were largely Asian or American Indian. Educational level ranged from two to 16 years of schooling, mean 10.6 years, median 11.0 years. The sample demographic characteristics (including the skewed racial/ethnic breakdown) reflect the demographic composition of the Cook County Jail.

Procedures

All detainees, excluding persons with gunshot wounds or other traumatic injuries, were part of the sampling pool. Personnel at the jail referred all persons targeted for participation in the project, regardless of their psychiatric morbidity, state of drug or alcohol intoxication, potential for violence, or fitness to stand trial. Subjects were selected via random numbers as they waited to be processed in the jail intake area. To ensure that the sample contained approximately equal numbers of misdemeanants and felons, the interviewers alternated between them in the sampling process.

Interviewers were clinical psychologists who had extensive training in interviewing techniques, psychopathology, and the data collection instrument. Interviewer consistency was scrupulously maintained after the initial three-month training period via mock interviews with live subjects, spot checks, and videotape training. Detainees who consented to be subjects were paid $5 which was mailed to an address of their choice. Of 767 subjects approached, 35 (4.6 percent) refused to participate. The low refusal rate was probably due to the detainees' viewing the research project as a way to avoid the crowded and dismal conditions of the regular intake area. Four other subjects were excluded: two "duplicate" subjects (they were re-arrested and randomly selected again) and two others who appeared to be confabulating their responses. Thus, the final N was 728.

Subjects were interviewed in a soundproof, private glass booth within the jail intake area using the National Institute of Mental Health Diagnostic Interview Schedule (NIMH-DIS).[34] Empirical tests have documented the reliability of the NIMH-DIS in both institutionalized samples and the general population[35–38] (in contrast, see the report by Anthony, *et al.*[39] The DIS systematically differentiates between disorders that were ever manifest, even if currently remitted (lifetime disorders), and disorders in which symptoms have been recently experienced (current disorders).

The NIMH-DIS provides diagnostic categories rather than global psychopathology scores. DSM-III diagnoses are scored from the interview data by a computer program written expressly for this purpose.[40] Because of subject variance over time and the rarity of many disorders, it is difficult to assess the reliability and validity of psychiatric assessment instruments such as the DIS.[41] Nevertheless, a reliability check of twenty pairs of interviews found 93 percent agreement across all diagnoses; 85 percent were given nearly identical profiles. The interview lasted between one and three hours, depending on the number of positive symptoms of the detainee.

The baseline (general population) data were obtained from the NIMH Epidemiologic Catchment Area program[42] which, also using the NIMH-DIS, calculated the prevalence rates of mental disorders in five cities: Baltimore (n = 3,481), New Haven (n = 5,034), Los Angeles (n = 3,131), Raleigh-Durham (n = 3,921), and St. Louis (n = 3,004).[5]

Because the jail sample was male, female subjects were eliminated from the comparison data. We also omitted all Hispanics (as well as other subjects who were neither Black nor White) from both data sets because there was an insufficient number of Hispanic jail detainees to include in the analysis (n = 47). Since the five-city sample excluded persons under the age of 18, we eliminated 53 subjects under that age from the jail sample in the comparison. Because almost all the jail sample were under age 60, we also excluded subjects over age 60 from the comparison. The final sample includes 627 jail detainees and 3,481 from the general population in the five cities: Baltimore, n = 825; New Haven, n = 748; Los Angeles, n = 431; Raleigh-Durham, n = 832; and St. Louis, n = 818. The complex sampling design of the five-city sample[43] required adjustment of the variances and standard errors presented in this paper using design effects estimated with Taylor Series Linearization provided by the National Institute of Mental Health.[44]

We calculated the prevalence rates of three disorders: major depressive episode, manic episode, and schizophrenia (including schizophreniform disorder). For each disorder, separate analyses were conducted to differentiate between "current" (defined as symptomatic within two weeks of the interview) and "lifetime" diagnoses. We conformed with the Epidemiologic Catchment Area study in that the disorders are not mutually exclusive: a subject could meet criteria for more than one disorder.[45]

In order to attain adequate statistical power for the jail-general population comparison, the data from the five cities needed to be combined. Before doing so, we compared the prevalence rates of each disorder across the five cities. This precautionary measure ensured that any observed differences between the jail and the general population data were not merely an artifact of the idiosyncratic characteristics of one or two of the cities. A loglinear analysis indicated that there were no significant or substantial dissimilarities in prevalence rates across the five cities (data available on request to author).

Results

Tables 1 and 2 show the prevalence rates with their standard errors, differences between the jail and the five-city sample, and the ratio of the differences in prevalence rates to the standard error of the difference (differences of proportions test[46]) for current and lifetime disorders, respectively. The uneven partitioning of age is dictated by the limited number of jail detainees over age 32 (n = 117). We did not simultaneously cross-classify by race and age because the resulting decrease in cell size would reduce power to an unacceptable level. The tables do not present data on socioeconomic status (SES) because the only SES indicators used in the five-city general population study (income and occupation) measure the individual's *achieved* status. These variables are inappropriate for this analysis because they have inconsistent bidirectional and temporal effects on the dependent variable. Low income and unemployment may either precede or result from severe mental illness.[47,48] Race is an indirect indicator of SES because of the strong correlation between these two factors.[49,50]

Tables 1 and 2 show that, overall, prevalence rates of current and lifetime major depression, mania, and schizophrenia, and "any severe disorder" (that is, any of the three

TABLE 1—Current Prevalence, Standard Errors, and Standard Errors of Differences for Major Depression, Mania, Schizophrenia and "Any Severe" Disorder for Jail and Five-City Samples

		Major Depression				Mania				Schizophrenia				Any Severe Disorder			
	N	Rate %	SE %	Diff. %	Diff./SE	Rate %	SE %	Diff. %	Diff./SE	Rate %	SE %	Diff. %	Diff./SE	Rate %	SE %	Diff. %	Diff./SE
All	4,281	1.49	0.36			0.30	0.19			1.18	0.32			2.50	0.47		
Non-Jail	3,654	1.07	0.22			0.12	0.08			0.91	0.22			1.84	0.31		
Jail	627	3.94	0.78	2.87	4.61**	1.36	0.46	1.24	4.48**	2.74	0.65	1.83	3.03**	6.36	0.98	4.52	5.33**
By race																	
Black	1,541	1.62	0.53			0.51	0.31			1.60	0.55			3.15	0.75		
Non-Jail	994	0.80	0.37			0.14	0.17			1.03	0.46			1.73	0.57		
Jail	547	3.11	0.74	2.31	3.12**	1.18	0.46	1.04	2.52*	2.63	0.68	1.60	2.01*	5.74	1.00	4.01	3.75**
White	2,740	1.42	0.62			0.19	0.32			0.95	0.43			2.14	0.69		
Non-Jail	2,660	1.17	0.27			0.12	0.10			0.87	0.26			1.89	0.37		
Jail	80	9.60	3.31	8.43	5.08**	2.55	1.77	2.43	3.81**	3.52	2.07	2.65	1.74	10.57	3.46	8.68	3.95**
By age (years)																	
18–22	706	0.82	0.60			0.11	0.24			1.90	0.91			2.61	1.06		
Non-Jail	511	0.43	0.38			0.00	—			1.51	0.77			1.94	0.85		
Jail	195	1.85	0.97	1.42	1.67	0.40	0.45	—	—	2.91	1.21	1.40	0.97	4.36	1.47	2.42	1.47
23–27	838	2.17	0.92			0.09	0.22			1.00	0.63			2.92	1.07		
Non-Jail	639	1.12	0.54			0.00	—			0.74	0.48			1.65	0.70		
Jail	199	5.56	1.63	4.44	3.35**	0.39	0.44	—	—	1.82	0.95	1.08	1.07	6.99	1.81	5.34	3.31**
28–32	736	2.14	1.06			0.56	0.66			1.72	0.96			3.70	1.39		
Non-Jail	619	1.29	0.59			0.08	0.16			1.17	0.61			2.36	0.85		
Jail	117	6.62	2.31	5.33	3.17**	3.10	1.61	3.02	3.81**	4.61	1.95	3.44	2.09*	10.80	2.88	8.44	3.64**
33–60	2,002	1.20	0.44			0.36	0.40			0.81	0.42			1.86	0.60		
Non-Jail	1,885	1.15	0.32			0.21	0.15			0.73	0.28			1.72	0.42		
Jail	117	2.01	1.30	0.86	0.65	2.85	1.54	2.64	3.65**	2.18	1.36	1.45	1.24	4.18	1.86	2.46	1.42

*Significant at .05.
**Significant at .01.

disorders) are significantly higher in the jail sample than in the five-city sample. The overall prevalence rates for current disorders are 1.24 percent to 4.52 percent higher among jail detainees than in the five-city sample; for lifetime disorders, the rates are 2.01 percent to 5.07 percent higher in the jail sample. In general, the differences in both current and lifetime prevalence rates between the jail and five-city samples hold when controlling for race. Except for current schizophrenia among Whites, and lifetime schizophrenia among both Whites and Blacks, the differences between the jail and the five-city sample remain statistically significant for Blacks and Whites, ranging from 1.04 percent (current mania among Blacks) to 8.43 percent (current major depression among Whites). The differences between the jail and the five-city sample are somewhat less robust when controlling for age, but this may reflect the small sample sizes.

The difference of proportions test (Tables 1 and 2) is less powerful with low prevalence rates than other techniques, e.g., loglinear analysis.[51] Given the limitations of the difference of proportions test, we reanalyzed the data using hierarchical loglinear modeling[52] (Table 3). This technique enables us to test the differences between the jail and five-city samples while holding both age and race constant.

Table 3 reports the parameters, the ratio of parameters to their standard errors (roughly equivalent to the likelihood ratio chi-square test), and model statistics for the final loglinear models for each current and lifetime diagnosis. Empty cells were set to .05, rather than the less conservative technique of setting them to structural zeros and adjusting degrees of freedom.[53] Notice that for each of the eight models, the site (jail/five-city sample) by diagnosis (present/absent) effect is retained and is in the hypothesized direction. In other words, we can reject the hypothesis of homogeneity for all current and lifetime disorders. In all cases, the coefficient for a diagnosis by site interaction exceeds its standard error by at least 2.58, or with a probability of less than .01. The analysis thus strongly supports our hypothesis: the prevalence rates of major depression, mania, and schizophrenic disorders are significantly higher in the jail than in the five-city sample, and are not an artifact of race or age differences between the two samples.

Discussion

This study provides reliable data concerning the relative prevalence of severe mental disorder among urban jail detainees in Cook County, Illinois, and the five-city Epidemiologic Catchment Area sample of the general population: for all race-age subgroups, the observed jail rates of schizophrenia, major depression, and mania were two to three times higher than in the general population. Moreover, these prevalence rates likely underestimate the true prevalence of mentally ill persons who are processed through the criminal justice system; samples obtained at the jail level omit all persons who are arrested but not incarcerated because they are diverted to a mental health facility during their pretrial hearing.

Our results do not enable us to ascertain whether mental disorder is a causal determinant of a criminal career, or merely a frequent trait among these offenders. Comparison with similar studies is of little utility. As demonstrated in the Appendix, their methodological limitations do not permit us to infer with any certainty whether our prevalence rates are higher than those found in prior studies. Our data thus provide necessary, but not sufficient, evidence that the mentally ill may be diverted into the criminal justice process.

In their exhaustive review of the literature, Monahan and Steadman[27] concluded that the apparently greater prevalence

TABLE 2—Lifetime Prevalence, Standard Errors, and Standard Errors of Differences for Major Depression, Mania, Schizophrenia and "Any Severe" Disorder for Jail and Five-City Samples

		Major Depression				Mania				Schizophrenia				Any Severe Disorder			
	N	Rate %	SE %	Diff. %	Diff./SE	Rate %	SE %	Diff. %	Diff./SE	Rate %	SE %	Diff. %	Diff./SE	Rate %	SE %	Diff. %	Diff./SE
All	4,281	3.53	0.50			0.64	0.27			1.99	0.40			5.15	0.63		
Non-Jail	3,654	3.15	0.37			0.32	0.14			1.70	0.30			4.41	0.47		
Jail	627	5.75	0.93	2.60	2.65**	2.51	0.63	2.19	5.26**	3.71	0.76	2.01	2.52*	9.48	1.17	5.07	4.09**
By race																	
Black	1,541	2.94	0.71			0.91	0.41			2.64	0.70			5.29	0.96		
Non-Jail	994	1.86	0.56			0.19	0.20			2.05	0.64			3.31	0.79		
Jail	547	4.90	0.92	3.04	2.99**	2.22	0.63	2.03	3.76**	3.70	0.81	1.65	1.57	8.89	1.22	5.58	4.00**
White	2,740	3.87	0.77			0.49	0.43			1.62	0.49			5.07	0.87		
Non-Jail	2,660	3.64	0.47			0.37	0.17			1.56	0.34			4.82	0.58		
Jail	80	11.54	3.59	7.90	2.84**	4.50	2.33	4.13	3.89**	3.52	2.07	1.96	0.98	13.49	3.84	8.67	2.55*
By age (years)																	
18–22	706	1.54	0.79			0.22	0.34			2.46	1.02			3.76	1.25		
Non-Jail	511	1.11	0.60			0.00	—			2.29	0.94			3.23	1.09		
Jail	195	2.66	1.16	1.55	1.28	0.80	0.64	—	—	2.91	1.21	0.62	0.37	5.16	1.59	1.93	0.96
23–27	838	5.17	1.31			0.40	0.44			1.38	0.72			6.42	1.50		
Non-Jail	639	4.28	1.04			0.00	—			1.21	0.82			5.28	1.23		
Jail	199	8.02	1.93	3.74	1.74	1.67	0.91	—	—	1.92	0.98	0.71	0.58	10.09	2.14	4.81	1.92
28–32	736	5.74	1.49			1.35	0.89			3.01	1.20			7.67	1.81		
Non-Jail	619	5.12	1.15			0.77	0.51			2.38	0.87			6.36	1.36		
Jail	117	9.04	2.66	3.92	1.36	4.44	1.91	3.67	2.56**	6.37	2.27	3.99	1.79	14.57	3.28	8.21	2.38*
33–60	2,002	2.74	0.63			0.62	0.52			1.70	0.64			4.19	0.91		
Non-Jail	1,885	2.68	0.48			0.36	0.20			1.47	0.39			3.80	0.61		
Jail	117	3.77	1.77	1.09	0.55	4.85	1.99	4.49	4.77**	5.44	2.11	3.97	2.38*	10.54	2.85	6.74	2.64**

*Significant at .05.
**Significant at .01.

of mental disorder among offenders disappears when sociodemographic factors are taken into account. In this study, however, the differences between the jail and general population persisted even after controlling for race and age.

It is also interesting to note that the observed ratio of current jail rates to current population rates is substantially higher than the comparable ratio of lifetime rates. This finding lends further support to the criminalization hypothesis because we know the arrest occurred during a period of active illness. Mentally ill persons with co-occurring substance abuse and personality disorders may be the most vulnerable to arrest because few treatment alternatives are available.**,[4] Clearly, further research is needed to disentangle the relationship between mental disorder and criminality, as well as to ascertain if mentally ill persons who are more appropriately treated within the mental health system are being funneled into the criminal justice system.

The finding that over 6 percent of all incoming jail detainees were suffering from a "current" psychotic illness suggests several public policy implications.

First, since disorders such as schizophrenia, major depression, and mania require immediate attention, jails must routinely screen all incoming detainees for severe mental disorder.[54] Interestingly, although the courts mandate that jails conduct routine mental health evaluations,[55–57] many jails do not do so.[58] The relative paucity of routine evaluation programs in jails is probably due to insufficient fiscal and mental health resources, as well as a dearth of screening instruments that are appropriate for the jail setting. Recent developments in screening techniques designed to be used within correctional settings[59] are likely to aid in the detection of mental illness. Jail administrators should incorporate these or similar instruments into their routine intake process to ensure that severely ill detainees will be detected and treated.

Second, the prevalence of detainees who are actively psychotic suggests that jail administrators must negotiate programmatic relationships with mental health facilities. Unfortunately, although the courts have stipulated that mentally ill prisoners must receive treatment for mental disorders,[60–62] many jails, particularly those in rural areas, have no mental health liaison.[63] Detainees can either be treated "in house," or referred outside.[64] Whichever system is used, it must be designed to minimize bureaucratic impediments to treatment.

Third, in accordance with the American Bar Association Mental Health Criminal Justice Standards,[65] mentally ill detainees who have committed minor crimes, e.g., trespassing and disorderly conduct, should be diverted to the mental health system. Clearly, persons whose offenses are more symptomatic of mental illness than of criminality should be treated as disordered rather than disorderly.

In sum, our results cannot be interpreted as evidence that the mentally ill are increasingly subject to incarceration. Nevertheless, the data document that the prevalence rate of severe mental disorder is significantly higher in a typical urban jail than in the general population. Additional epidemiological work is needed to ascertain the extent to which this finding is generalizable to other geographical areas. In accordance with court rulings, and in view of the number of severely disturbed detainees in our jails, we must allocate sufficient resources and develop and implement innovative treatment programs so that the mentally ill in jail may be treated expeditiously and in the most humane manner possible.

**Abram KM, Teplin LA: Co-occurring disorders among mentally ill jail detainees: Prevalence, patterns and implications. Manuscript submitted.

TABLE 3—Loglinear Modeling of Major Depressive, Mania, and Schizophrenia, and "Any Severe" Disorder with Site, Age, and Race

	Major Depressive				Mania				Schizophrenia				Any Severe Disorder			
	Current		Lifetime		Current		Lifetime		Current		Lifetime		Current		Lifetime	
	Coeff.	Coeff/SE	Coeff.	Coeff/SE	Coeff.	Coeff/SE	Coeff.	Coeff/SE	Coeff.	Coeff/SE	Coeff.	Coeff/SE	Coeff.	Coeff/SE	Coeff.	Coeff/SE
Site	0.64716	7.79429**	0.72082	9.59050**	0.36704	2.06367**	0.36565	2.90012**	0.69515	7.37676**	0.77727	9.78684**	0.66814	0.98984	0.73620	11.82454**
Age	−0.08916	−1.98927*	−0.49406	−3.41389**	−0.08916	−1.98927*	−0.68544	−1.82100	−0.08914	−1.98884*	−0.08914	−1.98884*	−0.08917	−1.98941*	−0.28710	−2.94520**
	0.03183	0.72611	0.22196	2.40217*	0.03183	0.72611	−0.21753	−0.76806	0.03183	0.72596	0.03183	0.72596	0.03165	0.72654	0.09918	1.27409
	−0.24995	−4.93573**	0.01302	0.13690	−0.24995	−4.93573**	0.24017	1.14957	−0.24995	−4.93586**	−0.24995	−4.93586**	−0.24995	−4.93585**	−0.07200	−0.90589
Race	0.23301	6.22200**	0.06238	0.89179	0.23301	6.22200**	0.23304	6.22283**	0.23302	6.22212**	0.23302	6.22212**	0.23298	6.22283**	0.14572	2.51508**
Diagnosis	1.93002	24.72801**	1.58786	23.86679**	2.74907	15.65617**	2.51726	15.29506**	2.06427	22.97210**	1.83130	24.68055**	1.66601	27.18680**	1.32669	26.86695**
Age by diagnosis			0.43504	3.02528**			0.60959	1.61575							0.22683	2.35182*
			−0.20492	−2.27764*			0.25906	0.91142							−0.07513	−0.99454
			−0.28878	−3.19583*			−0.50552	−2.40392*							−0.20997	−2.82254*
Race by diagnosis			0.18961	2.85341**											0.10255	1.95333
Site by age	−0.34099	−7.60792**	−0.34967	−7.76005**	−0.34099	−7.60792**	−0.35214	−7.80981**	−0.34101	−7.60835**	−0.34101	−7.60835**	−0.34098	−7.60778**	−0.35156	−7.79333**
	−0.23844	−5.43896**	−0.23417	−5.32806**	−0.23844	−5.43895**	−0.24658	−5.58891**	−0.23844	−5.43881**	−0.23844	−5.43881**	−0.23846	−5.43939**	−0.23556	−5.34996**
	0.01154	0.22779	0.01890	0.37175	0.01154	0.22779	0.02443	0.47626	0.01154	0.22791	0.01154	0.22791	0.01154	0.22791	0.02311	0.45316
Site by race	−0.72519	−19.36411**	−0.73429	−19.40516**	−0.72519	−19.36411**	−0.72522	−19.36494**	−0.72519	−19.36423**	−0.72519	−19.36423**	−0.72515	−19.36845**	−0.73223	−19.39179**
Site by diagnosis	0.33326	4.26966**	0.27160	3.66280**	0.60486	3.44474**	0.61423	4.97229**	0.27986	3.11444**	0.19861	2.67667**	0.32106	5.23927**	0.26795	4.48820**
Degree of freedom	20		16		20		17		20		20		16		16	
Model G^2	23.95568		10.54086		26.69138		14.87989		17.75165		20.08493		17.80153		11.33859	
Model Probability	0.244		0.837		0.144		0.604		0.604		0.452		0.600		0.788	

*Significant at .05.
**Significant at .01.

ACKNOWLEDGMENTS

This work was supported by Public Health Service Grant MH37988. I would like to thank Dr. Karen Mirsky Abram and Nancy Pruett for their insightful comments on an earlier version of this paper and Giovanni Moneta for his initial work on the analysis. Dr. Gary M. McClelland provided extensive advice and assistance in preparing and analyzing these data. Laura Coats was invaluable in the editing and preparation of the manuscript. Drs. Lee Robins and William Eaton, and Don Rae, MA, were instrumental in preparing the ECA data.

REFERENCES

1. Abramson MF: The criminalization of mentally disordered behavior. Possible side-effect of a new mental health law. Hosp Community Psychiatry 1972; 34:101–107.
2. Teplin LA: The criminalization of the mentally ill: Speculation in search of data. Psychol Bull 1983; 94:54–67.
3. Teplin LA: The Criminalization Hypothesis: Myth, Misnomer or Management Strategy. Rockville, MD: National Institute of Mental Health (in press).
4. Teplin LA: Criminalizing mental disorder: The comparative arrest rate of the mentally ill. Am Psychol 1984; 39:794–803.
5. Regier DA, Boyd JH, Burke JD, *et al:* One-month prevalence of mental disorders in the United States. Arch Gen Psychiatry 1988; 45:977–986.
6. Robins LN, Helzer JE, Weissman MM, *et al:* Lifetime prevalence of specific psychiatric disorders in three sites. Arch Gen Psychiatry 1984; 41:949–958.
7. Bolton A: A study of the need for and availability of mental health services for mentally disordered jail inmates and juveniles in detention facilities. Boston: Arthur Bolton Associates, 1976.
8. Petrich J: Rate of psychiatric morbidity in a metropolitan county jail. Am J Psychiatry 1976; 133:1439–1444.
9. Petrich J: Psychiatric treatment in jail: An experiment in health-care delivery. Hosp Community Psychiatry 1976; 27:413–415.
10. Piotrowski KW, Losacco D, Guze SE: Psychiatric disorders and crime: A study of pretrial psychiatric examinations. Dis Nerv System 1976; 3:309–311.
11. Swank GE, Winer DE: Occurrence of psychiatric disorder in a county jail population. Am J Psychiatry 1976; 133:1331–1333.
12. Kal EF: Mental health in jail. Am J Psychiatry 1977; 134:463.
13. Schuckit MA, Herrman G, Schuckit JJ: The importance of psychiatric illness in newly arrested prisoners. J Nerv Ment Dis 1977; 165:118–125.
14. Nielsen ED: Community mental health services in the community jail. Community Ment Health J 1979; 15:27–32.
15. Monahan J, McDonough LB: Delivering community mental health services to a county jail population. Bull Am Acad Psychiatry Law 1980; 8:28–32.
16. Whitmer GE: From hospitals to jails: The fate of California's deinstitutionalized mentally ill. Am J Orthopsychiatry 1980; 50:65–75.
17. Morgan C: Service delivery models: A summary of examples. *In:* Dunn D, Steadman H (eds): Mental health services in local jails: Report of a special national workshop. Crime and delinquency issues: A monograph series (DHHS Pub. No. ADM 82–1181). Washington, DC: Govt Printing Office, 1982.
18. Lamb HR, Grant RW: The mentally ill in an urban county jail. Arch Gen Psychiatry 1982; 39:17–22.
19. Lamb HR, Grant RW: Mentally ill women in a county jail. Arch Gen Psychiatry 1983; 40:363–368.
20. Ninzy NM: Mental health in an Ohio jail community. Ohio State Med J 1984; 80: 95–99.
21. Virginia Department of Mental Health and Mental Retardation and the Virginia Department of Corrections: The mentally ill in Virginia's jails. Final report of the Joint Task Force, 1984.
22. Glaser WF: Admissions to a prison psychiatric unit. Aust NZ J Psychiatry 1985; 19:45–52.
23. Guy E, Platt JJ, Zwerling I, Bullock S: Mental health status of prisoners in an urban jail. Crim Justice Behav 1985; 12:29–53.
24. Valdisserri EV, Carroll KR, Hartl AJ: A study of offenses committed by psychotic inmates in a county jail. Hosp Community Psychiatry 1986; 37:163–166.
25. Lazerwitz B: Sampling theory and procedures. *In:* Blalock HM Jr, Blalock AB (eds): Methodology in Social Research. New York: McGraw Hill, 1968.
26. US Department of Justice, Bureau of Justice Statistics: BJS Data Report, 1987 (NCJ-110643). Washington, DC: Dept of Justice, 1988.
27. Monahan J, Steadman HJ: Mentally Disordered Offenders. New York: Plenum Press, 1983.
28. US Department of Justice, Bureau of Justice Statistics: Special Report: The Prevalence of Imprisonment (NCJ-933657). Washington, DC: Dept of Justice, 1985.
29. US Department of Justice, Bureau of Justice Statistics: Jail inmates 1986 (Bulletin NCJ-107123). Washington, DC: Dept of Justice, 1987.
30. US Department of Justice, Bureau of Justice Statistics: Our crowded jails: A national plight (NCJ-111846). Washington, DC: Dept of Justice, 1988.
31. Zimmerman J, Keilitz I, Fitch W, *et al:* Forensic Mental Health Screening and Evaluation in Jails: Perspectives on Mental Health and the Law. Occasional Paper #2. Williamsburg, VA: National Center for State Courts, 1981.
32. US Department of Justice: 1988 Census of Jails. Bureau of Justice Statistics. Washington, DC: Govt Printing Office, 1990.
33. Cohen J: Statistical Power Analysis for the Behavioral Sciences, 2nd Ed. Hillsdale, NJ: Lawrence Erlbaum Associates, 1988; 52–56.
34. Robins LN, Helzer JE, Croughan J, Williams J, Spitzer R: NIMH Diagnostic Interview Schedule: Version III. Rockville, MD: National Institute of Mental Health, Division of Biometry and Epidemiology, 1981.
35. Robins LN, Helzer JE, Ratcliff K, Seyfried W: Validity of the Diagnostic Interview Schedule, Version II: DSM-III diagnoses. Psychol Med 1982; 12:855–870.
36. Helzer JE, Robins LN, McEvoy LT, *et al:* A comparison of clinical and Diagnostic Interview Schedule diagnoses. Arch Gen Psychiatry 1985; 42:657–666.
37. Robins LN, Helzer JE, Croughan J, Ratcliff K: National Institute of Mental Health Diagnostic Interview Schedule: Its history, characteristics and validity. Arch Gen Psychiatry 1981; 38:381–389.
38. Burke JD: Diagnostic categorization by the Diagnostic Interview Schedule (DIS): A comparison with other methods of assessment. *In:* Barrett J, Rose R (eds): Mental Disorders in the Community. New York: Guilford Press, 1986; 255–285.
39. Anthony JC, Folstein M, Romanoski AJ, *et al:* Comparison of the lay Diagnostic Interview Schedule and a standardized psychiatric diagnosis. Arch Gen Psychiatry 1985; 42:667–675.
40. Robins LN: DSM-III/DIS-IIIA Diagnostic Programs. St. Louis: Washington University, 1985.
41. Robins LN: Epidemiology: Reflections on testing the validity of psychiatric interviews. Arch Gen Psychiatry 1985; 9:918–924.
42. Eaton WW, Regier D, Locke B, Taube C: The Epidemiologic Catchment Area Program of the National Institute of Mental Health. Public Health Rep 1981; 96:319–325.
43. Holzer CE, Spitznagel E, Jordan KB, Timbers DM, Kessler LG, Anthony JC: Sampling the household population. *In:* Eaton WW, Kessler LG (eds): Epidemiologic Field Methods in Psychiatry. The NIMH Epidemiologic Catchment Area Program. New York: Academic Press Inc, 1985; 23–48.
44. Shah BV: SESUDAAN: Standard errors program for computing of standardized rates from sample survey data. Research Triangle Park, NC: Research Triangle Institute, 1981.
45. Boyd JH, Burke JD, Gruenberg E, *et al:* Exclusion criteria of DSM-III. Arch Gen Psychiatry 1984; 41:983–989.
46. Blalock HM Jr: Social Statistics. Rev 2nd Ed. New York: McGraw-Hill, 1979; 232–236.
47. Dohrenwend BP, Dohrenwend BS: Social Status and Psychological Disorder: A Causal Inquiry. New York: John Wiley & Sons, 1969.
48. Robins L: Social correlates of psychiatric illness: Can we tell causes from consequences? *In:* Redlich FC (ed): Social Psychiatry. Baltimore: Williams and Williams, 1969; 154–167.
49. Thomas ME, Hughes M: The continuing significance of race: A study of race, class and quality of life in America, 1972–1985. Am Soc Rev 1986; 51:830–841.
50. US Bureau of the Census: Statistical Abstract of the United States 1984 (104th Ed.) Washington, DC: Govt Printing Office, 1983.
51. Hanushek EA, Jackson JE: Statistical Methods for Social Scientists. Orlando, FL: Academic Press Inc, 1977; 188.
52. Haberman SJ: Analysis of Qualitative Data. Vol. 2. New York: Academic Press, 1978; 1–90.
53. Feinberg SE: The Analysis of Cross-classified Categorical Data. Cambridge, MA: MIT Press, 1980; 142–159.
54. Teplin LA: Detecting disorder: The treatment of mental illness among jail detainees. J Consul Clin Psychol v. 58, 1990.
55. *Alberti v Sheriff of Harris County*, 406 F Supp 649, 677 (SD Tex 1975).
56. *O'Bryan v County of Saginaw* 473 F Supp 582, 455 F Supp 437 (Mich 1978).
57. *Pugh v Locke*, 406 F Supp 318 (MD Ala 1976).
58. McCarty D, Steadman HJ, Morrissey JP: Issues in planning mental health services. Federal Probation 1982; December:56–63.
59. Teplin LA, Swartz J: Screening for severe mental disorder in jails: The development of the Referral Decision Scale. Law Hum Behav 1989; 13:1–18.
60. *Bowring v Godwin*, 551 F 2d 44 (4th Cir 1977).
61. *Estelle v Gamble*, 429 US 97 (1976).
62. *Jones v Witzenberg*, 323 F Supp 793 (ND Ohio 1971).
63. National Coalition for Jail Reform: Removing the chronically mentally ill from jail. Case studies of collaboration between local criminal justice and mental health systems. Washington, DC: National Coalition for Jail Reform and the National Institute of Mental Health, 1984.
64. Morgan C: Developing mental health services for local jails. Crim Justice Behav 1981; 8:259–273.
65. American Bar Association: The ABA Standards for Criminal Justice, 2nd Ed. Washington, DC: ABA, 1986.

APPENDIX

The Prevalence of Mentally Disordered Persons in Jails: A Summary of the Most Recent Research

Study	Description of Sample	Findings
Bolton[7] 1976	N = 1,084 (gender unknown) from 5 county jails	6.7% psychotic
Petrich[8] 1976	N = 539 referred for evaluation; 80% male; 42% felons	49% psychotic 10% depression
Petrich[9] 1976	N = 122 referred for evaluation; 84% male; 80% felons	29% schizophrenia 12% depression 6% mania
Piotrowski, Losacco, & Guze[10] 1976	N = 50 selected from those referred for evaluation; 86% male	22% schizophrenia 10% bipolar
Swank & Winer[11] 1976	N = 445 referred for evaluation; gender unknown; 41% felons N = 100 randomly selected; gender unknown; 71% felons	26% psychosis 5% psychosis
Kal[12] 1977	N unknown; random sample	Any DSM-III diagnosis:50% females; 63% males
Schuckit, Herrman & Schuckit[13] 1977	N = 100 males randomly selected; no felony convictions; no current drug charge	3% affective 48% any diagnosis
Nielson[14] 1979	N unknown; sample referred	24% psychosis
Monahan & McDonough[15] 1980	N = 632 referred for evaluation; 82% male; 55.5% misdemeanants	32% schizophrenia
Whitmer[16] 1980	N = 500 (gender unknown) "in need of treatment"	Averaged 3 prior psychiatric hospitalizations
Morgan[17] 1981–2	N unknown; sheriff's perceptions of mental illness	Estimates from 4–50%
Lamb & Grant[18] 1982	N = 102 males; random sample from referrals for evaluation; drug/alcohol excluded; 53% felons	75% schizophrenia 22% affective disorder
Lamb & Grant[19] 1983	N = 101 females, random sample from referrals for evaluation; drug/alcohol excluded; 37% felons	59% schizophrenia 35% affective disorder
Ninzy[20] 1984	N = 50 volunteers; 74% males	26% psychosis
Virginia DMH[21] 1984	N = 171 "mentally ill" as identified by staff	40% schizophrenia 21% mania
Glaser[22] 1985	N = 50 referred for evaluation; Australians (gender unknown)	48% schizophrenia 16% affective disorders
Guy, Platt, Zwerling & Bullock[23] 1985	N = 486 males randomly selected; 96 given diagnostic interview	12% schizophrenia 4% affective disorder
Valdiserri, Carroll, & Hartl[24] 1986	N = 769 referred for evaluation; 86% male	17% psychotic 5.5% predicted morbidity rate: total jail

Case-Control Study of Lung Cancer and Truck Driving in the Teamsters Union

N. Kyle Steenland, PhD, Debra T. Silverman, PhD, and Richard W. Hornung, PhD

Abstract: We conducted a case-control study of lung cancer deaths in the Teamsters Union to compare the risk of different occupations within the teamsters, after controlling for smoking and other confounders. Occupations with no presumed exposure to diesel fumes were used as the nonexposed group. The study population consisted of 996 cases and 1,085 controls who had died in 1982-83 after applying for pensions. Next of kin provided information on smoking, work history, and other potential confounders. Work history data were also obtained from the Teamsters Union. While no single job category had a significant excess risk compared to the non-exposed group, certain sub-groups were elevated. The odds ratio for those with long-term employment as long-haul truckers after 1959 (an approximate date for the introduction of diesel engines) was 1.55 (95% CI: 0.97, 2.47). Long-term drivers of primarily diesel trucks had an odds ratio of 1.89 (95% CI: 1.04, 3.42). Overall, our results suggest that diesel truck drivers have an excess risk of lung cancer compared to other teamsters in jobs outside the trucking industry. However, our findings were not uniformly consistent and our data have many limitations, the most important of which is the lack of data on exposure to diesel fumes. (*Am J Public Health* 1990; 80:670–674.)

Introduction

Inhaled diesel exhaust causes lung tumors in rodents,[1] and there is limited industrial hygiene data showing that truck drivers are exposed to diesel exhaust.[2] Recent human studies[3,4] also have indicated an excess lung cancer risk for diesel-exposed workers, after controlling for smoking. To test the hypothesis that truck drivers have an excess risk of lung cancer, we conducted a case-control study of lung cancer in the Teamsters Union. The risk for men in different occupations within the trucking industry was compared to that of men outside the trucking industry in jobs with no presumed diesel exposure. We had no direct data on level of exposure to diesel exhaust. A separate National Institute for Occupational Safety and Health/National Cancer Institute (NIOSH/NCI) project is now in progress to measure current exposure to diesel exhaust in the trucking industry, and to estimate historical exposures.

Methods

Study Population and Data Sources

Cases and controls were selected from the 10,699 male decedents from the Central States Teamsters files who had filed claims for pension benefits (requiring 20 years tenure in the union) and who had died in 1982 and 1983. We obtained death certificates for 10,485 (98 percent) of these men. Cases were all deaths from lung cancer, coded as ICD 162 or 163 for underlying or contributory cause on the death certificate (n = 1,288). Controls (n = 1,452) were every sixth death from the entire file (sorted by Social Security number), excluding deaths from lung cancer, bladder cancer, and motor vehicle accident.

Next of kin were questioned in detail on work history and potential confounders such as smoking, diet, and asbestos exposure. The response rate was 81 percent (see Figure 1). Most non-response was the result of our inability to locate the next of kin. Of the responses, 80 percent of the interviews were obtained by mail and 20% via follow-up phone calls; 76 percent were completed by spouses, and 24 percent were completed by some other next of kin. On the average, the next of kin had known the decedents for 39 years (38 for cases, 40 for controls).

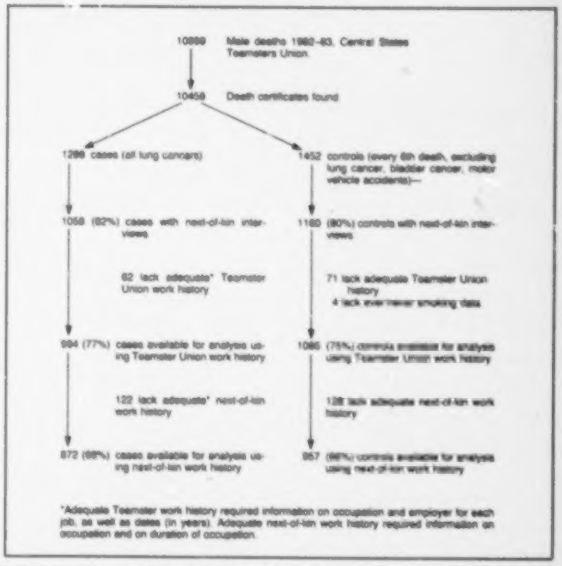

10699 Male deaths 1982-83, Central States Teamsters Union
10485 Death certificates found
1288 cases (all lung cancers)
1452 controls (every 6th death, excluding lung cancer, bladder cancer, motor vehicle accidents)—
1058 (82%) cases with next-of-kin interviews
1180 (80%) controls with next-of-kin interviews
62 lack adequate* Teamster Union work history
71 lack adequate Teamster Union history
4 lack ever/never smoking data
996 (77%) cases available for analyses using Teamster Union work history
1085 (75%) controls available for analyses using Teamster Union work history
122 lack adequate* next-of-kin work history
128 lack adequate next-of-kin work history
872 (68%) cases available for analysis using next-of-kin work history
957 (66%) controls available for analysis using next-of-kin work history
*Adequate Teamster work history required information on occupation and employer for each job, as well as dates (in years). Adequate next-of-kin work history required information on occupation and on duration of occupation.

FIGURE 1—Cases and Controls Use in Study of Lung Cancer and Truck Driving in the Teamsters Union

Men were then classified into the job category in which they had worked the longest (diesel truck driver, gasoline truck driver, driver of both types of trucks, truck mechanic, and dock worker), according to 1980 US Census occupation and industry codes.[5] Those who had never worked in any of these job categories were classified either as non-exposed, or as men who had held other jobs with potential diesel exposure (see Appendix A). Approximately 12 percent of the study subjects could not be classified into a principal job category because of missing data (e.g., the number of years worked in specific jobs was unknown to next of kin).

A second work history file was created based on the Teamster Union pension application which listed all teamster

---

Address reprint requests to N. Kyle Steenland, PhD, Industrywide Studies Branch, Division of Surveillance, Hazard Evaluations and Field Studies, National Institute for Occupational Safety and Health, Robert A. Taft Laboratories, 4676 Columbia Parkway, Cincinnati, OH 45226. Dr. Hornung is also with NIOSH; Dr. Silverman is with the National Cancer Institute. This paper, submitted to the Journal July 17, 1989, was revised and accepted for publication November 7, 1989.

jobs, as reported by the subject. Each job listed occupation, employer, and dates of employment. Both applicants and Teamster Union staff spent considerable effort to ensure the accuracy of these pension applications.

For the Teamster Union data, each job for each individual was assigned a three-digit US Census code (see Appendix A) for occupation and industry[5] (the latter obtained from commercial listings of employers which included type of industry). The four principal job occupations based on Teamster Union records were long-haul drivers, short-haul or city drivers, truck mechanics, and dock workers (see Appendix A). The Teamster Union work history did not provide information on whether men drove diesel or gasoline trucks. Men were again categorized according to the job category in which they had worked the longest. Most men had worked in only one exposed job category.

Diesel trucks were gradually introduced into the trucking fleet in the 1950s and 1960s in most of the country (before 1950s in Western states). Diesel trucks formed the majority of heavy duty truck sales for the first time in 1961.[6] However, many trucking companies (where most study subjects worked) had completed most of the dieselization of their fleets by 1960, while independent drivers and nontrucking firms may have obtained diesel trucks later (personal communication from companies in NIOSH/NCI industrial hygiene survey). We have chosen 1960 as the date used in our analysis, but we also report results using 1965 as the date for presumed dieselization.

Case-Control Analyses

The number of cases and controls available for analysis is shown in Figure 1. The method of analysis was unconditional logistic regression.[7] A series of dummy (0/1) variables was used to estimate the risk of a number of different job categories in relation to the same nonexposed group (see Appendix B).

All data on covariates, such as smoking and diet, were taken from the next-of-kin interview, while work history data were taken either from the Teamster Union pension applications or from next-of-kin interviews. Separate analyses were conducted for each type of work history data. Analyses considered employment (usual versus never) in a principal job category, as well as the number of years worked in that job. In some analyses using the Teamster Union work histories, only employment after 1959 (or 1964) was considered.

Details on covariate selection for inclusion in the model can be found in Appendix B. In summary, the only covariates that were included in the final model were age (five categories), smoking (six categories), asbestos (dichotomous), and a dichotomous variable for having held other jobs with potential diesel exposure. The fit of this basic model and four continuous exposure variables from the Teamster Union work history was evaluated via the method described by Lemeshow and Hosmer.[8] The chi square resulting from this test was 18.0, with 18 degrees of freedom (p = .46), indicating a good fit of the model.

Results

Table 1 shows the number of men and the average duration of employment in the different job categories. Table 2 shows the results of analyses when exposure was considered as a dichotomous variable (i.e., main job versus nonexposed), based on Teamster Union and on next-of-kin work history. No single job category had a highly elevated risk compared to non-exposed men, although most were somewhat elevated.

We further analyzed the Teamster Union work history data by estimating risk by duration of employment as a categorical variable (Table 3). When no cutoff date for employment was used, there was little indication of any trend of increasing risk with increasing duration of employment for any of the job categories. When only employment after 1959 was considered, both long-haul (test for trend, p = .04) and short-haul drivers (test for trend, p = .22) showed an increase in risk with increased years of exposure; truck mechanics showed no increase in risk with increased years of exposure; truck mechanics showed no increase in risk with time. If 1964 was used for a cutoff date instead of 1959, long-haul drivers continued to show a significant positive trend (p = .04), and the highest duration category (13+ years) had an odds ratio of 1.64 (95% CI: 1.05, 2.57). Short-haul drivers, however, no longer showed a positive trend. If duration of employment was used as a continuous variable, significant positive trends

TABLE 1—Number of Men and Average Years Worked in Different Job Categories by Case-Control Status, from Teamster Union Work History and Next-of-Kin Data

Teamster Union Data	Number Cases	Number Controls	Average Years in Job Cases	Average Years in Job Controls
Overall	994	1085	na	na
Long-haul truck driving	609	604	24.2	24.0
City truck driving	121	134	23.4	24.5
Truck mechanic	50	37	22.3	23.3
Dock worker	70	92	23.1	23.3
Other potentially exposed jobs	99	143	na	na
No potential for diesel exposure	45	75	na	na
Next-of-kin data				
Overall	994	1085	na	na
Primarily diesel truck driver	176	155	29.6	27.6
Primarily gasoline truck driver	245	309	29.0	29.7
Drove both types of truck	248	246	31.2	31.2
Diesel truck mechanic	43	41	32.4	32.3
Dock worker	28	40	25.4	25.1
Other potentially exposed jobs	87	128	na	na
No potential for diesel exposure	45	75	na	na
Missing required data	122		na	na

TABLE 2—Odds Ratios for Exposure Considered as Main Job (ever vs never)

Job category	Odds ratio* (95% CI)
Teamster Data	
Long haul driver	1.27 (.83–1.93)
Short haul driver	1.31 (.81–2.11)
Truck mechanic	1.69 (.92–3.09)
Dock worker	0.92 (.55–1.55)
Other potentially diesel exposed	1.44 (.88–2.39)
Next-of-kin data	
Truck driver, primarily diesel truck	1.42 (.89–2.26)
Truck driver, primarily gasoline truck	1.22 (.79–1.88)
Truck driver, both types of truck	1.25 (.81–1.95)
Truck mechanic (diesel)	1.35 (.74–2.47)
Dock worker	0.93 (.49–1.78)
Other potentially diesel exposed	1.54 (.93–2.15)

*Adjusted for age, smoking, and asbestos, the reference group is the non-exposed.

were observed both for long-haul drivers and mechanics, but only after 1959.

Similar trend analyses, based on next-of-kin data, are shown in Table 4. None of the job categories showed a significant increasing trend, although men whose main job was driving diesel trucks did show a marginal increase in risk with increasing duration of employment as a truck driver (p = .12). For truck drivers who drove 35 years or longer and drove primarily diesel trucks, the odds ratio for lung cancer was 1.89 (95% CI: 1.04, 3.42). If duration was considered as a continuous variable, only the trend for diesel truck drivers approached significance (p = .09).

Using either the Teamster Union work history or that supplied by next of kin, there were no significant interactions between age and exposure, or smoking and exposure. It did not appear that excess risks were confined to any specific category of smokers or non-smokers. The results for lagged analyses (discounting any exposure in the five years preceding death) were similar to the analyses with no lag.

Concordance between Teamster and next-of-kin job categories could not be easily evaluated because job categories were defined differently in each data set (e.g., diesel truck drivers based on next of kin, long-haul drivers based on Teamster Union data). Discrepancies between next-of-kin and Teamster Union work history could occur because next of kin were reporting lifetime work history, while the Teamster records only covered Teamster years. However, 90 percent of men identified by next of kin as primarily diesel truck drivers were long-haul drivers by Teamster Union data, and the corresponding proportions were 82 percent for mechanics and 81 percent for dock workers.

Discussion

The Teamsters Union keeps very thorough records regarding their members, which have not yet been fully utilized by epidemiologists. We were able to study Teamster pensioners using data on their vital status and work history which had been collected by the Union's Pension Fund. It is worth noting that the Union, jointly with employers, has created a Health and Welfare Fund which might be used for studying morbidity.

We observed positive trends in lung cancer risk with duration of employment for long-haul truck drivers after either 1959 or 1964 (Teamster work history), and for truck drivers who drove primarily diesel trucks (next-of-kin work

TABLE 3—Odds Ratios According to Length of Employment for Long-haul Truck Drivers, Short-haul Truck Drivers, and Truck Mechanics

Job Category	Length of Employment (years)	Odds Ratio (95% CI)*	Cases	Controls
No Cutoff Date				
Long-haul driver	1–21	1.25 (.78, 1.97)	205	218
	22–27	1.12 (.72, 1.77)	199	195
	28 or more	1.47 (.94, 2.31)	199	195
Short-haul driver	1–21	1.52 (.86, 2.71)	52	52
	22–27	1.73 (.92, 3.25)	40	34
	28 or more	0.83 (.45, 1.56)	29	57
Truck mechanic	1–21	2.23 (.97, 5.17)	22	13
	22–27	1.20 (.53, 2.72)	17	17
	28 or more	1.88 (.66, 5.35)	11	8
*Exposure after 1959***				
Long-haul driver	1–11	1.08 (.68, 1.70)	162	230
	12–17	1.41 (.90, 2.21)	228	203
	18 or more	***1.55 (.97, 2.47)	213	171
Short-haul driver	1–11	1.11 (.61, 2.03)	36	58
	12–17	1.15 (.63, 2.43)	37	45
	18 or more	1.79 (.94, 3.42)	40	31
Truck mechanic	1–11	1.83 (.80, 4.19)	19	16
	12–17	2.08 (.78, 5.52)	15	8
	18 or more	1.50 (.59,3.40)	16	13

*These data reflect two analyses, with employment restricted to after 1959 or without such a restriction. Each analysis included all the above exposure variables. Odds ratios were also adusted for age, smoking, and asbestos. Variables for work as a dock worker or in other potentially diesel-exposed jobs were also included in the model. For all comparisons, the non-exposed were those who had never worked in principal jobs categories nor in any other job potentially exposed to diesel exhaust.

**For the 1959 cutoff, we have used different cutpoints, because the maximum possible length of employment in a given job category is 23 years (1960–83). Cutpoints were chosen a priori by dividing long-haul drivers into three groups of approximately equal size. See text for results using 1964 cutoff.

***The linear trend in odds ratios for long-haul drivers was significant (p = .04). The coefficient was 0.027, which increased to .037 when exposure was defined after 1964.

SOURCE: Teamster Union Work History.

TABLE 4—Odds Ratios According to Length of Employment for Men as a Truck Driver or Mechanic for Men Whose Main Job Was Diesel Truck Driver, Gasoline Truck Driver, Driver of both Gasoline and Diesel Trucks, or Truck Mechanic

Main Job Category	Years of Employment* (years)	Odds Ratio (95% CI)**	Cases	Controls
Diesel truck driver	1–24	1.27 (.70, 2.27)	48	52
	25–34	1.26 (.74, 2.16)	72	67
	35 or more	1.89 (1.04, 3.42)	56	36
Gasoline truck driver	1–24	1.24 (.74, 2.16)	72	87
	25–34	1.10 (.67, 1.80)	87	112
	35 or more	1.34 (.81, 2.22)	86	111
Drove both truck types	1–24	1.27 (.71, 2.26)	50	51
	25–34	1.15 (.70, 1.90)	95	100
	35 or more	1.34 (.81, 2.20)	102	95
Truck mechanic	1–24	1.69 (.61, 4.67)	11	9
	25–34	1.39 (.63, 3.07)	20	16
	35 or more	1.09 (.44, 2.66)	12	16

*Years of employment as a truck driver does not necessarily signify years of employment driving a particular type of truck.

**All the above exposure variables were included in the model together. In addition, odds ratios were adjusted for age, smoking, and asbestos. Dock workers and men in other potentially exposed jobs were also included in model. For all comparisons, the non-exposed were those who had never worked in principal job categories nor in any other jobs potentially exposed to diesel exhaust.

SOURCE: Next-of-Kin Data

history). These findings suggest that there may be a real increase in risk with increased employment driving a long-haul or diesel truck.

Mechanics showed marginal elevated risks of lung cancer, especially based on Teamster work history. Preliminary sampling results from NIOSH/NCI surveys indicate that mechanics are likely to have been exposed to relatively high levels of diesel exhaust, especially in winter. However, the risk for mechanics did not appear to increase consistently with duration of employment. Also, there is evidence in the literature that mechanics have been exposed to low levels of asbestos in the course of working on brakes.[9] While we included data from next of kin on asbestos exposure in the model, the data on asbestos may have been inadequate to control for confounding.

Dock workers showed a slight deficit in risk. It is possible that dock workers have had less diesel exposure than truck drivers and mechanics. Dock workers frequently unloaded trailers disconnected from the cabs with engines in open air terminals. Short-haul drivers in the Teamster data, but not gasoline drivers (next of kin data), showed some evidence of increased risk.

The principal limitation in this study is the lack of exposure data indicating level of diesel exhaust for the different job categories studied. Such data would enable us to draw firmer conclusions with respect to the role of diesel exhaust, rather than employment in presumably diesel-exposed jobs. Unfortunately, little data exist on historical levels of diesel exhaust in the trucking industry.

Other limitations include: 1) possible misclassification of smoking habits by next of kin, 2) misclassification of exposure by next of kin, 3) a relatively small non-exposed group (n = 120) which by chance might have had a low lung cancer risk, and 4) lack of sufficient latency (time since first exposure) to observe a lung cancer excess. On the other hand, next-of-kin data on smoking have been shown to be reasonably accurate[10], non-differential misclassification of exposure (between cases and controls) would only bias our findings toward the null hypothesis of no association, and the trends of increased risk with increased duration of employment in certain jobs would persist even if the non-exposed group had had a higher lung cancer risk. Finally, the lack of potential latency would also make any positive results more striking.

In view of the above limitations, the results of this study should be considered cautiously. However, other recent findings support our results. The study of Boffetta, *et al*,[3] based on data from the American Cancer Society, indicates that truck drivers show an increased risk of lung cancer after correction for smoking (RR = 1.24, 95% CI: 0.93, 1.66), and that this increase is primarily associated with truck drivers with long duration of diesel exposure (self-reported). Other recent data support an increased lung cancer risk due to diesel exposure in general. The Boffetta study cited above showed an increased in lung cancer risk for those with diesel exposure in any occupation (RR = 1.18, 0.97–1.44), and this risk increased with duration. Garshick, *et al*,[4] conducted a case-control study of 1,256 lung cancer cases and matched controls who had died in 1982–83 after railroad work. Industrial hygiene assessments were done to determine which job categories had significant exposure to diesel exhaust. Those men with 20 years or more duration of employment in diesel-exposed jobs after 1959 (date when locomotives were all presumed to be diesel) showed a significant excess risk (odds ratio = 1.41) compared to the non-exposed, after controlling for smoking and asbestos. These positive findings for truck drivers and other diesel-exposed workers lend plausibility to our own results.

ACKNOWLEDGMENTS

The NIOSH clerical staff, led in this study by Pauline Bishak, and the Central States Teamsters Pension Fund staff (particularly Alan Levy, Mary Balint, Viola Baba, and Mark Felke) performed invaluable service in collecting the raw data for this study. A preliminary version of this paper was presented at the American Public Health Association conference in Chicago, October 25, 1989.

REFERENCES

1. Ishinishi N, Koizumni A, McClellan R, *et al*, (eds): Carcinogenic and Mutagenic Effects of Diesel Engine Exhaust. New York: Elsevier, 1986.
2. Ziskind R, Carlin T, Ballas J: Evaluating gas hazards inside heavy duty diesel truck cabs. Proceedings American Chemical Society, 4th Joint Conference on Sensing of Environmental Pollutants 1978; 377–383.
3. Boffetta P, Stellman S, Garfinkel L: Diesel exhaust exposure and mortality among males in the American Cancer Society Prospective Study. Am J Ind Med 1988; 14:403–415.

4. Garshick E, Schenker M, Munoz A, *et al*: A case-control study of lung cancer and diesel exposure in railroad workers. Am Rev Respir Dis 1987; 135:1242–1248.
5. US Census Bureau: Alphabetical Index of Industries and Occupations. Washington, DC: Govt Printing Officer, 1981.
6. Motor Vehicle Manufactures Association: Facts and Figures. Detroit, MI: The Association, 1962
7. SAS Institute: Supplemental Library Users Guide, Version 5 Edition. Cary, NC: SAS Institute, 1986.
8. Lemeshow S, Hosmer D: A review of goodness of fit statistics for use in the development of logistic regression models. Am J Epidemiol 1982; 115:92–106.
9. Rohl A, Langer A, Wolff M, *et al*: Asbestos exposure during brake liningmaintenance and repair. Environ Res 1976; 110–128.
10. McLaughlin K, Dietz E, *et al*: Reliability of surrogate information of cigarette smoking by type of informant. Am J Epidemiol 1987; 126:144–146.
11. Rothman K: Modern Epidemiology. Boston: Little, Brown, and Company, 1986.
12. Byers T, Vena J, Mettline C, *et al*: Dietary Vitamin A and lung cancer risk: Am J Edpidemiol 1984; 120:769–776.
13. Humble C, Samet J, Skipper B: Use of quantified and frequency indices of Vitamin A intake in a case-control study of lung cancer. Int J Epidemiol 1987; 16:341–346.

APPENDIX A

1980 Census[5] Occupation and Industry Codes Used in Defining Job Categories in Teamster Union Work Histories

Job category	Occupation Codes	Industry Codes
Long-haul driver	804 (truck driver, heavy)	any
City driver	805 (truck driver, light)	any
	806 (driver/sales for bakeries dairies, cleaners, or beverage company	111, 101. 771, 120, any if occupation is sales or route driver
Truck mechanic	507 (truck mechanic)	410 (trucking)
Dock worker	343 (checker)	410
	359, 363 (dispatcher)	410
	364 (shipping clerk)	410
	365 (stock clerk)	410
	368 (checker)	410
	843 (dock foreman)	410
	883 (dockman)	410
	856 (forklift operator)	410
	889 (warehouseman)	410
	859 (moving man)	410
Other jobs with potential diesel exposure	all except above jobs in 410 (trucking)	410
	503–517 (other mechanics)	any
	803–859 (other vehicle drivers)	any
	343 (checker)	any except 410
	359, 363 (dispatcher)	any except 410
	364 (shipping clerk)	any except 410
	365 (stock clerk)	any except 410
	368 (checker)	any except 410
	843 (dock foreman)	any except 410
	883 (dockman)	any except 410
	856 (forklift operator)	any except 410
	889 (warehouseman)	any except 410
	859 (moving man)	any except 410
	885 (service station work)	any
	019 (Teamster official)	410

APPENDIX B

Definition of Non-exposed and Selection of Covariates for Inclusion in the Model

The same non-exposed group was used for the analyses based on Teamster Union work history and the analyses based on next-of-kin work history. The non-exposed group was chosen from those who, based on Teamster work history, had never worked as either a long-haul driver, a short-haul driver, a mechanic, a dock worker, or any other potentially diesel-exposed job (Appendix A). Many of these men worked in dairies. Those with no potential diesel exposure via Teamster work history (n = 156) were then further reviewed to exclude anyone with any jobs reported by next of kin which might have had potential diesel exposure (e.g., men who drove trucks prior to joining the Teamsters Union and working in a non-exposed job). The classification of men into the non-exposed group, like all classifications into job categories, was done without any knowledge of case-control status.

Exposure variables and covariates were tested by adding them to a basic model, which included variables for smoking (six categories) and age (five categories). The odds ratios for lung cancer did not increase monotonically with age (using the youngest group as referent), prompting our use of categorical rather than continuous age variables (odds ratios, in reference to age less than 45 years, were 2.86 for ages 45–54, 3.42 for ages 55–64, 3.71 for ages 65–74, and 2.12 for ages greater than 75). Cigarette smoking was categorized into never smoker, current smoker (current at time of death) with unknown amount, current moderate smoker (one pack or less), current heavy smoker (more than one pack), former smoker who quit before 1963, and former smoker who quit 1963–79. The odds ratios for smokers, in reference to never smokers, were 5.64 for current light smokers, 8.85 for current heavy smokers, 4.48 for former smokers who quit in the 20 years before death, and 2.41 for former smokers who quit prior to 20 years before death.

Variables other than exposures, smoking, or age were not included in the model if they did not act as confounders (did not change the estimates of exposure effect), and if they did not interact significantly with the exposure variables. Trends with duration of employment were tested either via inclusion of continuous variables, or via categorization of duration and a test for trend described by Rothman.[11] Cutoff points for categories of duration were chosen a priori, based on dividing long-haul (or diesel when using next-of-kin data) truck drivers into three categories of duration with approximately equal numbers in each. These same cutpoints were then used for the other job categories.

The following variables were neither significant predictors of lung cancer nor acted as confounders: smoking a pipe or cigar, race, state of residence at death, coffee drinking, work in construction, work in shipyards, and driving a truck with a partner who smoked. The frequency of consumption of certain types of vegetables and fruits was of interest because of a possible protective effect of Vitamin A.[12,13] The only significant diet variable was consumption of deep yellow vegetables. The odds ratio for infrequent consumers (one or two times a month or less) of yellow vegetables was 1.38 (1.10–1.76), and for average consumers (one or two times per week) was 1.26 (.98–1.62), both compared to frequent consumers (three or more times per week). However, these variables did not confound the associations under study and were not included in the final model. Asbestos exposure was a significant predictor (odds ratio = 2.80), had some confounding effect on the exposure variables for mechanics, and was retained in the final model.

47

Nongenetic Influences of Obesity on Other Cardiovascular Disease Risk Factors: An Analysis of Identical Twins

BETH NEWMAN, PHD, JOSEPH V. SELBY, MD, CHARLES P. QUESENBERRY, JR., PHD, MARY-CLAIRE KING, PHD,
GARY D. FRIEDMAN, MD, AND RICHARD R. FABSITZ, MA

Abstract: The importance of genetic influences on obesity has been emphasized recently. We conducted matched co-twin analyses of 250 pairs of White, male, monozygotic twins from the National Heart, Lung, and Blood Institute (NHLBI) Twin Study. Entirely in the absence of genetic influences, obesity was significantly associated with systolic and diastolic blood pressures; one-hour, post-load glucose; total, LDL-, and HDL-cholesterol; and triglycerides among these 42–55 year old men. Similar results were obtained in longitudinal analyses of weight change during adulthood (from mean age of 20 to mean age of 48 years) and risk factor status at middle-age. These results indicate that behaviors and environmental exposures that occur later in life are responsible, at least in part, for the associations between adult obesity and cardiovascular disease risk, supporting the appropriateness of weight reduction efforts during adulthood. (*Am J Public Health* 1990; 80:675–678.)

Introduction

Obesity, hypertension, glucose intolerance, and high lipid levels tend to cluster in families.[1,2] Obesity is also correlated with these other cardiovascular disease risk factors in individuals.[3,4] The extent to which this clustering is caused by common genetic influences on multiple risk factors or by shared environmental or behavioral factors is unknown. In a previous report,[5] investigators from the National Heart, Lung, and Blood Institute (NHLBI) Twin Study demonstrated that many cardiovascular disease risk factors have genetic determinants. Using another analytic approach with the same data, we sought to determine whether associations exist between obesity and the other cardiovascular disease risk factors independent of genetic influences.

Comparison within genetically identical, monozygotic twin pairs removes all genetic variability.[6] If intrapair differences in cardiovascular disease risk factors are related to intrapair differences in obesity, these associations must be attributable to environmental or behavioral, and therefore potentially alterable, influences. In this paper, we use longitudinal data from a group of White, male, monozygotic twins in a matched-pair analysis to assess the nature of the relationships of obesity and adult weight gain with blood pressure, glucose tolerance, and lipid levels at middle-age.

Methods

Study Subjects

Data collection for the NHLBI Twin Study took place between July 1969 and June 1973 in five locations: Framingham, Massachusetts; Indianapolis, Indiana; and Los Angeles, San Francisco, and Davis, California. Participants were 514 pairs of White male twins who were members of the National Academy of Sciences-National Research Council Twin Panel[7] and who lived in California, New England, or within 200 miles of Indianapolis. All twins were born during the years 1917 through 1927 and served in the US Armed Forces during the period from World War II through the Korean War. Twins were ages 42–55 years at the study examination. In this report, only the 250 monozygotic twin pairs were included.

Data Collection

After an overnight fast, both members of each twin pair received physical examinations on the same day by different physicians. Weight and height were measured in pounds and inches. Each individual's weight and height also were obtained from records of the physical examination prior to military induction (ages 17 to 28). After conversion to kilograms and meters, body mass index (BMI) was calculated as the measure of obesity (weight/height^2). Blood pressure was measured three times using a standard sphygmomanometer: first by a nurse, later twice by a physician. The participant was seated, and both systolic and diastolic (Phase V) blood pressures were recorded. The mean of the two physician readings was used in this report. Blood samples were collected to determine glucose tolerance, plasma cholesterol and triglycerides, and serotypes for assignment of zygosity. A 50-g oral glucose load was given one hour before blood was drawn unless the subject had diabetes and was currently using insulin or oral hypoglycemic agents or had glycosuria of 3+ or greater. Glucose levels were measured at the examination centers using an SMA 12/60 instrument (Technicon Instruments, Tarreytown, NY). Total, HDL-, and LDL-cholesterol[8] and triglyceride[9] levels were determined at three regional laboratories; specimens were exchanged to maintain standardization. Additional details of the recruitment process, response rates, zygosity determination, clinical examination, and questionnaire are available.[5,10]

Statistical Analyses

Matched-pair t-tests and linear regression models were computed using SAS computer programs.[11] Both members of a twin pair were included in both the unmatched and matched analyses. However, unmatched analyses were adjusted to avoid overestimating the statistical significance of parameter estimates due to correlated errors between co-twins.[12] For the matched analyses, regression models incorporated intrapair differences of the dependent and independent variables.

From the Division of Research, Kaiser Permanente Medical Care Program, Oakland, CA (Newman, Selby, Quesenberry, Friedman); University of California-Berkeley, School of Public Health (Newman, King); and the Clinical and Genetic Epidemiology Branch, National Heart, Lung, and Blood Institute, Bethesda, MD (Fabsitz). Address reprint requests to Beth Newman, PhD, Division of Research, Kaiser Permanente Medical Care Program, Northern California Region, 3451 Piedmont Avenue, Oakland, CA 94611. This paper, submitted to the Journal August 28, 1989, was revised and accepted for publication December 7, 1989.

Editor's Note: See also related editorial p 657 this issue.

The intrapair difference for each variable was obtained by subtracting the value for one twin from that of the other in each monozygotic twin pair. For analyses of weight change, the value of BMI at the military induction physical examination was subtracted from the value of BMI at the study examination.

Sample sizes varied between 219 and 245 pairs in the different regression analyses because of missing values and specific exclusions of individuals using medications that affect blood pressure, glucose, or lipid levels. Because the distribution of triglycerides was skewed toward higher values, we used the natural log transformation on this variable in all regression analyses.

Results

Means and standard deviations for obesity, weight change, and the other cardiovascular disease risk factors for the twins as individuals are presented in Table 1.

Within monozygotic twin pairs, heavier twins had significantly higher levels of systolic and diastolic blood pressures, glucose (one-hour, post-load), total cholesterol, LDL-cholesterol, and triglycerides and lower levels of HDL-cholesterol than their genetically identical, lighter-weight brothers (Table 2). Regression coefficients measuring the association of obesity with each risk factor are indicated in Table 3. Examination BMI was significantly associated with all the cardiovascular disease risk factors in the unmatched analyses, independently of age. Matched-pair regression analyses revealed even stronger, significant, cross-sectional relationships between BMI and the cardiovascular disease risk factors. Only the regression coefficient for HDL-cholesterol was somewhat reduced.

Obesity was then partitioned into two variables—BMI at military induction (average age 20 years), and change in BMI during the 20 to 34 years between the induction and study examinations. The independent influences of the two obesity parameters on the other cardiovascular disease risk factors are indicated in Table 4. In both the unmatched and matched analyses, weight gain during adulthood was significantly associated with all the cardiovascular disease risk factors measured. In addition, induction BMI was independently and significantly related to systolic and diastolic blood pressures, total cholesterol, and triglycerides in the matched analyses. The negative regression coefficient between change in BMI and HDL-cholesterol was the only coefficient smaller in the matched analysis than in the unmatched analysis; the inverse relationship between induction BMI and HDL-cholesterol in the unmatched analysis was reduced and no longer statistically significant in the matched analysis. The proportion of nongenetic variance explained by both measures of obesity in the matched analyses ranged from 4 percent for HDL-cholesterol to 10 percent for total cholesterol, 11 percent for systolic blood pressure, and 13 percent for diastolic blood pressure and triglycerides.

TABLE 1—Obesity Measures at Military Induction and at Study Examination and Cardiovascular Disease Risk Factors at Study Examination (1969–73) in a Sample of White, Male, Monozygotic Twins

Variables	Number of Individuals	Mean ± Standard Deviation
Body mass index (kg/m^2)		
at military induction	499	21.9 ± 2.4
at study examination	499	25.7 ± 3.2
change between induction and examination	498	3.8 ± 2.9
Age at study examination (years)	500	47.8 ± 3.1
Systolic blood pressure (mmHg)	439	127.0 ± 16.3
Diastolic blood pressure (mmHg)	439	81.3 ± 10.2
One-hour, post-load glucose (mg/dl)	465	162.6 ± 51.9
Total cholesterol (mg/dl)	491	219.7 ± 35.0
HDL-cholesterol (mg/dl)	486	44.2 ± 12.8
LDL-cholesterol (mg/dl)	485	143.5 ± 33.5
Triglycerides (mg/dl)	490	136.1 ± 89.2

TABLE 2—Mean Levels of Cardiovascular Disease Risk Factors at Ages 42–55 Years Comparing Heavier and Lighter Brothers within Monozygotic Twin Pairs

Variables	Heavier Twin	Lighter Twin	Mean Intrapair Difference (95% CI)
Body mass index (kg/m^2)	26.6	24.8	1.8 (1.6, 2.0)
Systolic blood pressure (mmHg)	129.6	124.5	5.1 (3.1, 7.1)
Diastolic blood pressure (mmHg)	82.7	80.0	2.7 (1.4, 3.9)
One-hour, post-load glucose (mg/dl)*	161.7	156.0	5.6 (0.3, 11.2)
Total cholesterol (mg/dl)*	222.6	217.1	5.5 (1.9, 9.1)
HDL-cholesterol (mg/dl)*	43.1	45.3	−2.2 (−3.5, −1.0)
LDL-cholesterol (mg/dl)*	145.7	141.6	4.2 (0.8, 7.5)
Triglycerides (mg/dl)*	147.6	124.9	22.7 (12.5, 33.0)

*Conversion factor to SI Units in mmol/L:
Glucose—0.05551
Cholesterol (total, HDL, LDL)—0.02586
Triglycerides—0.01129

TABLE 3—Regression Coefficients+ (± standard errors) for Cross-sectional Relationships between Body Mass Index (BMI) and other Cardiovascular Disease Risk Factors in Middle-Aged, White, Male Monozygotic Twins

Dependent Variables	Unmatched Analysis	Matched Analysis
Systolic blood pressure	1.47 ± 0.28	2.23 ± 0.43
Diastolic blood pressure	1.06 ± 0.17	1.44 ± 0.26
One-hour, post-load glucose	3.95 ± 0.80	4.79 ± 1.22
Total cholesterol	2.43 ± 0.51	3.86 ± 0.74
HDL-cholesterol	−0.97 ± 0.18	−0.83 ± 0.27
LDL-cholesterol	1.13 ± 0.49	2.37 ± 0.71
Ln triglycerides	0.065 ± 0.008	0.073 ± 0.013

+Coefficients are age-adjusted: age was included in the unmatched regression models, but was not related significantly to any outcome; matched co-twin analyses control for age in that co-twins are perfectly matched on age.

Discussion

In response to the recent emphasis on the importance of genes in influencing adult obesity,[13–15] we used data from identical twins to address the following question: do nongenetic differences in obesity contribute to levels of blood pressure, glucose intolerance, and lipids? The results of our analyses indicate that significant associations exist between obesity and many cardiovascular disease risk factors entirely in the absence of genetic influences. These results were obtained from both cross-sectional analyses of obesity and concurrent risk factor status and longitudinal analyses of change in weight during adulthood and risk factor status during middle-age. Furthermore, because matched-pair analyses also control for any factors that are identical or very similar between co-twins, such as the influences of shared early environment and culture, the observed associations

TABLE 4—Regression Coefficients[+] (±standard errors) for Longitudinal Relationships between Cardiovascular Disease Risk Factors and Body Mass Index (BMI) from Military Induction to Middle-age in White, Male, Monozygotic Twins

Dependent Variables	Unmatched Analysis		Matched Analysis	
	Change in BMI	Induction BMI	Change in BMI	Induction BMI
Systolic blood pressure	1.66 ± 0.31	1.06 ± 0.39	2.27 ± 0.44	1.93 ± 0.79
Diastolic blood pressure	1.18 ± 0.19	0.79 ± 0.24	1.48 ± 0.26	1.09 ± 0.47
One-hour, post-load glucose	4.60 ± 0.87	2.19 ± 1.21	4.99 ± 1.25	3.22 ± 2.29
Total cholesterol	2.83 ± 0.55	1.33 ± 0.77	3.89 ± 0.76	3.80 ± 1.37
HDL-cholesterol	−0.97 ± 0.20	−0.97 ± 0.28	−0.84 ± 0.27	−0.75 ± 0.50
LDL-cholesterol	1.34 ± 0.54	0.54 ± 0.76	2.35 ± 0.72	2.55 ± 1.33
Ln triglycerides	0.075 ± 0.008	0.041 ± 0.011	0.075 ± 0.013	0.057 ± 0.023

[+] Coefficients are age-adjusted: age was included in the unmatched regression models, but was not related significantly to any outcome; matched co-twin analyses control for age in that co-twins are perfectly matched on age.

may be attributed more specifically to environmental exposures or personal behaviors of adulthood.

These results do not preclude genetic influences on obesity, blood pressure, glucose tolerance, or lipid levels.[5,16] Nor do they reveal the specific, nongenetic mechanisms underlying the associations of cardiovascular disease risk factors with obesity. Obesity may serve as a marker of an atherogenic lifestyle, or it may contribute directly to the increases in blood pressure, glucose intolerance, and lipid levels through mechanical or biochemical pathways. Regardless of the mechanisms involved, the effects observed here are not pleiotropic effects of genes regulating adult fatness. The generalizability of these findings is supported by the comparability of the twins participating in this study to White men in the general population[17–19] and to participants in similar studies.[20–23]

Weight change during adulthood was generally more important than baseline BMI at military induction in predicting cardiovascular disease risk factor status in the longitudinal analyses. However, for blood pressure, total cholesterol, and triglycerides, BMI in early adulthood also contributed independently to risk factor status in middle-age. When analyses for all cardiovascular disease risk factors were conducted including both examination BMI (middle-age) and induction BMI (early adulthood) in the models, only the examination BMI was statistically significant (data not shown). These results suggest that the attained level of obesity is important in elevating a person's risk for diabetes and cardiovascular disease, although duration of obesity may also play an independent role.

Although the influence of obesity on the other cardiovascular disease risk factors appears to be limited to less than 15 percent of the nongenetic variability of each risk factor, a significant, if moderate, effect of weight gain during adulthood was observed on each risk factor. The average man in our cohort was 69 inches tall and gained 28 pounds between age 20 and age 48. By middle-age, he had higher levels of each of the cardiovascular disease risk factors studied, except for HDL-cholesterol, than men who gained less weight. Associated with his weight gain were excesses of 9.5 mmHg systolic blood pressure, 6.2 mmHg diastolic blood pressure, 21.0 mg/dl glucose (one-hour, post-load), 16.4 mg/dl total cholesterol, 9.8 mg/dl LDL-cholesterol, and a decrease of 3.5 mg/dl HDL-cholesterol. Similarly, his triglyceride level was 40 percent higher than that of men who did not gain weight between military induction and middle-age; this represents roughly a 50 mg/dl excess. These excesses occurred independent of initial BMI and can be attributed solely to nongenetic influences on the basis of the matched co-twin analysis (Table 4). The cumulative impact of similar or larger weight gains on multiple risk factors clearly contributes significantly to an increase in cardiovascular disease risk.

The causes of adult obesity are complex and undoubtedly involve both genetic and nongenetic influences.[24,25] Bouchard, *et al*,[26] have shown that monozygotic twins generally gain similar amounts of weight as a result of similar diets that include excess calories. However, unrelated twin pairs respond very differently to the same diet. This interpair variability is a reflection of gene-environment interaction, where an individual's response depends both on genetic predisposition as well as specific environmental exposure. Moreover, BMI is only a surrogate measure of obesity and may not capture the most relevant aspect related to cardiovascular disease risk. Unfortunately, more detailed measures of total body fat or fat distribution were not available from the induction or 1969–73 study examinations.

Despite the etiologic complexity of adult obesity, and the well-documented difficulty in its control, our findings provide some reason for optimism. They indicate that behaviors and environmental exposures that occur later in life are responsible, in part, for the association between adult obesity and cardiovascular disease risk factor status. They emphasize the importance of attained weight in influencing that risk, thereby suggesting that weight reduction will be effective even at later ages, when most individuals tend to gain weight. Thus, they confirm the clinical impression that weight control and prevention of obesity are likely to be successful in reducing a person's risk of cardiovascular disease and diabetes.

ACKNOWLEDGMENTS

This research was supported by Contract N01-HC-55028 from the National Heart, Lung, and Blood Institute.

REFERENCES

1. Perkins KA: Family history of coronary heart disease: Is it an independent risk factor? Am J Epidemiol 1986; 124:182–194.
2. King MC, Lee G, Spinner NB, *et al:* Genetic epidemiology. Ann Rev Public Health 1984; 5:1–52.

3. Hubert HB: The importance of obesity in the development of coronary risk factors and disease: The epidemiologic evidence. Ann Rev Public Health 1986; 7:493–502.
4. Burton BT, Foster WR, Hirsh J, Van Itallie TB: Health implications of obesity: An NIH Consensus Development Conference. Int J Obes 1985; 9:155–170.
5. Feinleib M, Garrison RJ, Fabsitz R, *et al:* The NHLBI Twin Study of Cardiovascular Disease Risk Factors: Methodology and summary of results. Am J Epidemiol 1977; 106:284–295.
6. Gesell A: The method of co-twin control. Science 1942; 95:446–448.
7. Jablon S, Neel JV, Gershowitz H, *et al:* The NAS-NRC Twin Panel: Methods of construction of the panel, zygosity diagnosis, and proposed use. Am J Hum Genet 1967; 19:133–161.
8. Manual of Laboratory Operations, Lipid Research Clinics Program. DHEW Pub. No. (NIH) 75-628. Washington, DC: Govt Printing Office, 1974.
9. Kessler G, Lederer H: Fluorometric measurement of triglyceride. *In:* Skeggs LI Jr, *et al* (eds): Automation in Analytic Chemistry: Technicon Symposia, 1965. New York: Mediad, 1966.
10. Feinleib M, Christian JC, Borhani NO, *et al:* The National Heart and Lung Institute Twin Study of Cardiovascular Disease Risk Factors: Organization and methodology. Acta Genet Med Gemellol (Roma) 1976; 25:125–128.
11. SAS User's Guide: Statistics, Version 5 Edition. Cary, NC: SAS Institute, Inc., 1985.
12. Fabsitz R, Feinleib M, Hubert H: Regression analysis with correlated errors: An example from the NHLBI Twin Study. J Chronic Dis 1985; 38:165–170.
13. Stunkard AJ, Sorensen TIA, Hanis C, *et al:* An adoption study of human obesity. N Engl J Med 1986; 314:193–198.
14. Stunkard AJ, Foch TT, Hrubec Z: A twin study of human obesity. JAMA 1986; 256:51–54.
15. Price RA, Cadoret RJ, Stunkard AJ, Troughton E: Genetic contributions to human fatness: An adoption study. Am J Psychiatry 1987; 144:1003–1008.
16. Austin MA, King MC, Bawol RD, *et al:* Risk factors for coronary heart disease in adult female twins: Genetic heritability and shared environmental influences. Am J Epidemiol 1987; 125:308–318.
17. Simopoulos AP, Van Itallie TB: Body weight, health, and longevity. Ann Intern Med 1984; 100:285–295.
18. National Center for Health Statistics: Blood pressure levels of persons 6–74 years, United States, 1971–1974. Vital Health Stat 11, No. 203, 1977.
19. National Center for Health Statistics: Total serum cholesterol levels of adults 18–74 years, United States, 1971–1974. Vital Health Stat 11, No. 205, 1978.
20. Borkin GA, Sparrow D, Wisniewski C, Vokonas PS: Body weight and coronary disease risk: Patterns of risk factor change associated with long-term weight change, The Normative Aging Study. Am J Epidemiol 1986; 124:410–419.
21. Khoury P, Morrison JA, Mellies MJ, Glueck CJ: Weight change since age 18 in 30- to 55-year-old Whites and Blacks. JAMA 1983; 250:3179–3187.
22. Gillum RF, Taylor HL, Brozek J, *et al:* Blood lipids in young men followed 32 years. J Chronic Dis 1982; 35:635–641.
23. Ashley FW, Kannel WB: Relation of weight change to changes in atherogenic traits: The Framingham Study. J Chronic Dis 1974;27:103–114.
24. Bouchard C, Perusse L, Leblanc C, *et al:* Inheritance of the amount and distribution of human body fat. Int J Obesity 1988; 12:205–215.
25. Price RA, Stunkard AJ: Comingling analysis of obesity in twins. Hum Hered 1989; 39:121–135.
26. Bouchard C, Tremblay A, Depres J-P, *et al:* Sensitivity to overfeeding: The Quebec experiment with identical twins. Prog Food Nutr Sci 1988; 12:45–72.

$3 Million Awarded to Public Health Schools

Almost $3 million in fiscal 1990 capitation grant funds were awarded by the Health Resources and Services Administration to 24 schools of public health.

The funds represent general institutional support which a school may expend for any purpose it judges will most effectively advance its educational program.

The grant recipients and the amounts awarded are:

- University of Alabama at Birmingham, $81,448;
- University of California, Berkeley, $172,670;
- Loma Linda University, California, $131,946;
- University of California, Los Angeles, $206,553;
- San Diego State University, California, $72,000;
- Yale University, New Haven, CT, $77,865;
- University of South Florida, Tampa, $60,272;
- University of Hawaii, Honolulu, $94,154;
- University of Illinois, Chicago, $71,674;
- Tulane University, New Orleans, LA, $124,127;
- Johns Hopkins University, Baltimore, MD, $223,494;
- University of Massachusetts, Amherst, $74,607;
- Boston University, Massachusetts, $76,887;
- Harvard University, Boston, MA, $152,471;
- University of Michigan, Ann Arbor, $224,472;
- University of Minnesota, St. Paul, $107,512;
- Columbia University, New York, NY, $127,059;
- University of North Carolina, Chapel Hill, $201,992;
- University of Pittsburgh, PA, $105,883;
- University of Puerto Rico, San Juan, $120,543;
- University of Oklahoma, Oklahoma City, $86,335;
- University of South Carolina, Columbia, $87,313;
- University of Texas, Houston, $155,729; and
- University of Washington, Seattle, $79,494.

More information is available from the Division of Associated and Dental Health Professions, Bureau of Health Professions, Room 8-101, 5600 Fishers Lane, Rockville, MD 20857, tel: 301-443-6853.

3T

The Differential Effect of Traditional Risk Factors on Infant Birthweight among Blacks and Whites in Chicago

JAMES W. COLLINS, JR., MD, MPH, AND RICHARD J. DAVID, MD

Abstract: We analyzed 103,072 White and Black births in Chicago from the 1982 and 1983 Illinois vital records, using 1980 median family income of mother's census tract as an ecologic variable. Thirty-one percent of Blacks and 4 percent of Whites resided in census tracts with median family incomes ≤$10,000/year. Only 2 percent of Black mothers, compared to 16 percent of White mothers, lived in areas where the median family income was greater than $25,000/year. Among Blacks with incomes ≤$10,000/year, maternal age, education, and marital status had minimal predictive power on the incidence of low birthweight (LBW) infants. Among high-risk mothers in the poorest areas the proportion of LBW infants in Blacks and Whites was less divergent than in higher income areas. Independent of residential area, low-risk Whites had half the occurrence of LBW infants as Blacks. We conclude that the extremes of residential environments show dramatic racial disparity in prevalence, yet the few low-risk Blacks still do less well than low-risk Whites. Traditional risk factors do not completely explain racial differences in neonatal outcome. (*Am J Public Health* 1990; 80:679–681.)

Introduction

Despite the dramatic improvements in perinatal care during the past 30 years, Black infants are twice as likely as White infants to die during their first month of life.[1–3] This is primarily related to the high incidence of low birthweight (<2500 grams) infants among Black mothers.[4–6] Cities such as Washington, DC, Detroit, and Chicago with large Black subpopulations have the highest neonatal mortality rates (NMR) in the country and are in part responsible for the United States' relatively poor international ranking in neonatal survival.[7] The mechanisms underlying this well-documented phenomenon are not well understood.

Low maternal socioeconomic status (SES) worsens pregnancy outcome.[8,9] Black mothers are more likely to be young, unmarried, poorly educated, and receiving welfare support.[8] However, Spurlock, *et al*,[10] found that poor and non-poor Black infants in Kentucky had no significant difference in birthweight distribution. There is limited available information on mothers of high SES, especially among ethnic groups with widespread poverty. Shiono, *et al*,[5] reported that in a middle class community Black mothers still had a high incidence of LBW infants. Similarly, Kleinman and Kessel[11] found that the ratio of low birthweight for Blacks versus Whites was actually higher among college educated mothers than for women who did not complete high school. Another recent study indicated that a 5 percent racial difference in low birthweight persisted independent of maternal residence.[12] Few studies have addressed the possibility that mature, educated, Black mothers who reside in high income communities might be less likely to deliver low birthweight infants.

For these reasons, we utilized Illinois vital records and US census income data to determine the extent to which disparities in traditional risk factors affect racial differences in pregnancy outcome in Chicago. We particularly wanted to learn if low maternal risk status reduces the number of low birthweight Black infants.

Methods

We analyzed all Black and White births in Chicago using a data set created by appending survival data from infant birth-death tapes as previously described[13] and income characteristics of mother's census tract (from 1980 census tapes) to records in the 1982 and 1983 birth files. Because of their strong association with neonatal outcome maternal age, education, and marital status were used as proxies of individual risk status. Median family income of mother's census tract residence was used as the primary proxy for environmental deterioration. Percent of families within each census tract living below the poverty level was used as an additional ecologic variable.

Among Blacks and Whites we calculated the proportion (per 100 live births) of LBW infants by census tract income, maternal age, and education for the entire study population. We stratified the study population by median family income of mother's census tract to examine the Black-White differential in residential environments. Within each grouped income area, we calculated the frequency of individual and ecologic risk variables. To explore the independent association of residential environment and pregnancy outcome, we determined the relation of maternal age, education, marital status, parity, and history of prior infant death to Black and White infant birth weight.

Relative risk with 95% confidence intervals are presented. Confidence limits were estimated by the Taylor Series method.[14]

Results

There were 51,827 Black and 51,245 White births in Chicago during 1982 and 1983. Black neonatal mortality was twice as high as that of Whites (16/1,000 vs 7/1,000) and the LBW proportions were twice as high in Blacks (14 percent vs. 6 percent). Only 2 percent of Black mothers, compared to 16 percent of White mothers, resided in census tracts in which the median family income was greater than $25,000/year. Conversely, 31 percent of Blacks lived in census tracts in which the average household income was less than $10,000/year; only 4 percent of White mothers resided in such impoverished neighborhoods (Figure 1).

The risk of LBW infants among Blacks remained essentially twice as high as that of Whites across all maternal income, education, and age groups. Although the higher

---

From the Department of Pediatrics, Northwestern University Medical School (Collins), and the Division of Perinatal Epidemiology, Cook County Hospital (David). Address reprint requests to James W. Collins, Jr, MD, MPH, Division of Neonatology, Children's Memorial Hospital, 2300 Children's Plaza, Chicago, IL 60614. This paper, submitted to the Journal July 25, 1989, was revised and accepted for publication December 20, 1989.

Editor's Note: See also related editorial p 653 this issue.

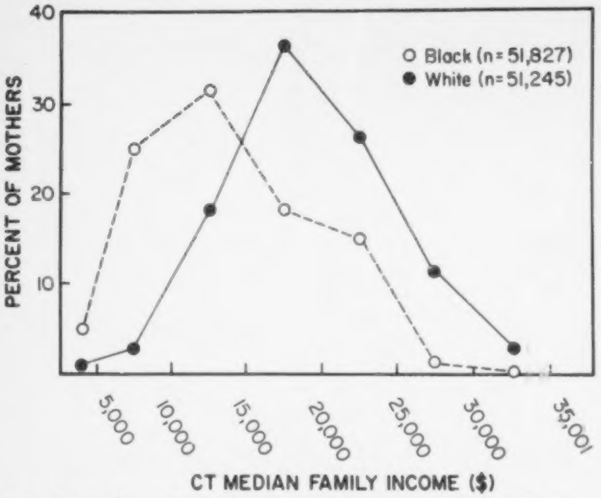


FIGURE 1—Census Tract Median Family Income Distribution of Black and White Mothers

Black risk appeared to be eliminated at very high incomes, the confidence intervals were broad because of the small number of Blacks (N = 200, or 0.4 percent) residing in census tracts with median family income over $40,000/year (Table 1).

Table 2 illustrates the racial distribution of risk factors by census tract income. White mothers were often Hispanic in low-income areas. Among mothers who resided in very low-income census tracts (≤$10,000/year) Blacks were almost 3.5-times as likely as Whites to reside in a census tract where more than one-half of the census tract cohabitants lived below the federally mandated poverty level (RR = 3.47, 95% CI 3.07, 3.92).

The differential effect of race on the LBW rates in low-income areas is apparent with maternal age. Among Black mothers who resided in higher income communities, the high proportion of LBW was minimally reduced and remained twice that of Whites independent of maternal education, age, and marital status (Table 3).

TABLE 1—Proportion of Low Birthweight (<2500 grams) and Relative Risk in Black and White Infants according to Income, Maternal Education, and Age

Variables	% LBW		Black Relative Risk (95% CI)
	Black	White	
Income (per year)			
≤$10,000	15	8	1.92 (1.64, 2.26)
$10,001–$20,000	14	6	2.12 (2.01, 2.24)
$20,001–$30,000	12	6	2.16 (1.99, 2.39)
$30,001–$40,000	10	5	2.26 (1.39, 3.54)
>$40,000	4	4	.98 (.13, 7.29)
Maternal Education (years)			
<12	16	7	2.40 (2.26, 2.55)
12	14	7	2.13 (1.93, 2.42)
13–15	12	5	2.14 (1.93, 2.42)
16	10	5	2.13 (1.76, 2.57)
≥17	9	5	1.87 (1.36, 2.53)
Maternal Age (years)			
≤19	14	8	1.44 (1.37, 1.55)
20–35	14	6	2.41 (2.30, 2.52)
>35	14	8	1.98 (1.64, 2.39)

TABLE 2—Distribution of Individual and Environmental Risk Factors by Census Tract Average Annual Income and Race

Variables	≤$10,000		$10,001–$20,000		$20,001–$30,000	
	Black	White	Black	White	Black	White
Age (years)						
% ≤19	34	19	27	3	22	8
Education (years)						
% <12	54	55	38	60	23	21
Marital Status						
% Unmarried	82	36	71	88	59	12
Maternal Ethnic Origin						
% Hispanic	<1	25	<1	23	<1	2
Poverty Level*	43	13	1	0	0	0

*Incidence rate of residing in a census tract in which more than 50% of families have annual incomes below the federally mandated poverty level.

Discussion

We found that high-risk Whites and Blacks had less divergent LBW rates in the poorest areas than in higher income areas while low-risk Whites had half the occurrence of LBW infants as Blacks regardless of the income of the area in which they lived. In Chicago, the percentages of Black and White mothers at the extremes of residential environment are dramatically different. Contrary to Spurlock, *et al*,[10] who reported that poor and non-poor Black infants in Kentucky had no difference in the incidence of LBW, we found that among all mothers, Black and White, low income is associated with a greater risk of low birthweight.

A wider Black-White gap at the upper end of the socioeconomic spectrum has been described in earlier studies such as that by Kleinman and Kessel,[11] but this could have been an artifact of the differences in the economic value of education (the usual proxy for income) between Blacks and Whites in the US. For example, the income disparity between college educated Blacks and Whites is greater than it is for those who did not finish high school.[15] Notwithstanding the limitations of the available grouped income data, our results confirm the results of Kleinman and Kessel and highlight the difficulty in defining a truly low-risk group of Black mothers using traditional risk factors.

Our data suggest that residential environment is an important risk factor that researchers need to take into account when examining the relation between race and neonatal outcome. Residing in a very low-income urban neighborhood is such a strong proxy of low birthweight for Blacks that traditional indicators of favorable outcome (education, age, marital status) fail to identify clearly a low risk subgroup. The intense concentration of extreme poverty combined with the related issues of disintegrating social networks, substance abuse, poor nutrition, smoking, and inadequate prenatal care may produce such a powerful negative force that isolated changes in the classical risk factors do not dramatically reduce the high percentage of low birthweight infants.

Racial differences in intrauterine growth rates exist.[16,17] However, when Wilcox and Russell controlled for Black-White disparities in birthweight distribution, the excess in mortality among Black infants was not eliminated.[17] The study found that a high incidence of small births among Blacks accounted for the majority of Black-White differences in survival.

The mechanisms of racial disparity in health in this

TABLE 3—Proportion of Low Birthweight (<2500 grams) Infants by Census Tract Average Annual Income and Race

Variables	<$10,000			$10,001–$20,000			$20,001–$30,000		
	Black (N = 15,889)	White (N = 1,851)	Black RR (95% CI)	Black (N = 27,754)	White (N = 27,867)	Black RR (95% CI)	Black (N = 8,936)	White (N = 20,199)	Black RR (95% CI)
					%LBW				
Maternal Age (years)									
≤19	14	13	1.02 (.78, 1.35)	14	8	1.72 (1.53, 1.93)	13	5	2.70 (2.15, 3.40)
20–35	16	7	2.40 (1.97, 2.93)	14	6	2.24 (2.10, 2.39)	12	6	2.20 (2.02, 2.41)
Maternal Education (years)									
≤12	16	9	1.85 (1.56, 2.21)	15	7	2.08 (1.97, 2.21)	14	6	2.21 (2.01, 2.43)
>12	12	5	2.43 (1.54, 3.82)	11	5	2.08 (1.80, 2.40)	11	5	2.14 (1.87, 2.46)
Marital Status									
Unmarried	16	11	1.20 (.96, 1.44)	15	9	1.76 (1.51, 2.05)	14	8	1.75 (1.51, 2.05)
Married	14	6	2.29 (1.80, 2.40)	11	6	2.00 (1.84, 2.18)	10	5	1.87 (1.67, 2.09)
Parity*									
Primaparity	13	8	1.64 (1.27, 2.12)	9	7	1.27 (1.14, 1.41)	12	6	1.99 (1.75, 2.74)
Low parity	14	6	2.44 (1.83, 3.25)	14	6	2.28 (2.11, 2.47)	13	5	2.62 (2.35, 2.93)
High parity	18	9	2.08 (1.53, 2.84)	18	8	2.39 (2.17, 2.64)	15	6	2.31 (1.94, 2.20)
Prior Infant Death									
Yes	22	9	6.37 (1.62, 25.10)	22	8	2.31 (1.82, 2.95)	25	5	3.94 (2.47, 6.89)
No	15	8	1.78 (1.52, 2.09)	14	6	2.15 (2.04, 2.27)	13	4	3.40 (3.11, 3.73)

*Parity was defined as high in third or higher numbered births to women under 25 years of age and fourth or higher numbered births to women 25 to 29 years of age. All other multiparous births were considered to represent low parity.[11]

society are complex and deep-rooted.[8,18] We suspect that the persistently high rate of low birthweight infants among Blacks reflects generations of poverty.[19] While the vital records-based studies can identify factors that affect perinatal outcome, there is a need for studies in greater depth. A limitation of the present study is that even with household income data for each birth we could not truly have "controlled" for Black-White economic differences since income cannot be equated with purchasing power.[20] Moreover, despite treating census tract income as an indicator of the environment within which people live, the lack of homogeneity among mothers of the same grouped income area limits the generalizability of the study.[21] A multidisciplinary approach is needed to better elucidate and remove causes of Black-White differences in neonatal outcome.

In summary, the extremes of residential environment show dramatic racial disparity in prevalence, yet traditional risk factors fail to explain why non-poor Blacks remain at high risk for poor pregnancy outcome. Given that a large percentage of Blacks in this country live at or below the poverty level we need to eliminate economic disparities between the races in addition to providing planned intervention if we hope to see the health gap closed. We suspect that it may require more than one generation of non-poverty and adequate services to see an impact upon infant birthweight.[19]

ACKNOWLEDGMENTS

Presented in part at the annual meeting of the American Pediatric Society and the Society for Pediatric Research, Washington, DC, 1988, and the annual meeting of the American Academy of Pediatrics, San Francisco, 1988.

REFERENCES

1. Binkin NJ, Rust KR, Williams RL: Racial differences in neonatal mortality: What causes of death explain the gap. Am J Dis Child 1988; 142:434–440.
2. Binkin NJ, Williams RC, Hogue CR, *et al:* Reducing Black neonatal mortality: Will improvement in birth weight be enough? JAMA 1985; 253:372–375.
3. Wise PH, First LR, Lamb GA, *et al:* Infant mortality increase despite high access to tertiary care: An evolving relationship among infant mortality, health care, and socioeconomic change. Pediatrics 1988; 81:542–548.
4. McCormick MC: The contribution of low birth weight to infant mortality and childhood morbidity. N Engl J Med 1985; 312:82–90.
5. Shiono PH, Klebanof MA, Graubord BI, Berendes HW, Rhoads GG: Birth weight among women of different ethnic groups. JAMA 1986; 255:48–52.
6. Institute of Medicine, Committee to Study the Prevention of Low Birth Weight: Preventing Low Birth Weight. Washington, DC: National Academy Press, 1985; 30–102.
7. Wegman ME: Annual summary of vital statistics–1987. Pediatrics 1988; 82:817–827.
8. Liberman E, Ryan K, Monson R, *et al:* Risk factors accounting for racial differences in the rate of premature birth. N Engl J Med 1987; 317:743–748.
9. Paneth N, Wallenstein S, Kielly J, Susser M: Social class indicators and mortality in low birth weight infants. Am J Epidemiol 1982; 116:364–375.
10. Spurlock CW, Hinds MW, Skaggs JW, Hernandez CE: Infant death rates among poor and non-poor in Kentucky, 1982 to 1983. Pediatrics 1987; 80:262–269.
11. Kleinman JC, Kessel SS: Racial differences in low birth weight. N Engl J Med 1987; 317:749–753.
12. Gould JB, Leroy S: Socioeconomic status and low birth weight: A racial comparison. Pediatrics 1988; 82:896–904.
13. Holland S, David R: An outcome coded birth file from machine readable vital records. Comput Biol Med 1985; 15:25–39.
14. Kleinbaum PG, Kupper LL, Morgenstern H: Epidemiologic Research—Principles and Quantitative Methods. New York: Van Nostrand Reinhold Company, 1982.
15. US Bureau of the Census: Statistical Abstract of the United States: 1987 (107th edition). Washington, DC: Dept of Commerce, 1986.
16. Collins JW, David RJ: Differential survival rates among low birth weight black and white infants in a tertiary care hospital. Epidemiology 1990; 1:16–20.
17. Wilcox AJ, Russell IT: Birthweight and perinatal mortality. III. Towards a new method of analysis. Int J Epidemiol 1986; 15:188–196.
18. Wenneker MB, Epstein AM: Racial inequalities in the use of procedures for patients with ischemic heart disease in Massachusetts. JAMA 1989; 261:253–257.
19. Baird D: Perinatal mortality. Lancet 1969; 1:511–515.
20. Centerwall BS: Race, socioeconomic status, and domestic homicide, Atlanta, 1971–72. Am J Public Health 1984; 74:813–815.
21. Morgenstern H: Use of ecologic analysis in epidemiologic research. Am J Public Health 1982; 74:1336–1344.

2T

The Dramatic Increase in the Rate of Low Birthweight in New York City: An Aggregate Time-Series Analysis

THEODORE JOYCE, PHD

Abstract: This paper uses monthly time-series data from 1968 through 1988 to test whether the observed increase in the incidence of low birthweight in New York City represents a reversal of the downward trend that existed prior to 1984. We find that the trend has shifted for both Whites and Blacks. We estimate that between July 1984 and December 1988 there were 3,110 additional low-birthweight births to Blacks and 1,385 additional low-birthweight births to Whites above what would have been expected had the rate of low birthweight not shifted upwards. The incremental costs of treating these infants in their first year of life is estimated at over $22,000,000 in 1986 dollars. (*Am J Public Health* 1990; 80:682–684.)

Introduction

The proportion of low birthweight births among Blacks in New York City has increased dramatically since 1984 and less dramatically among Whites (see Figure 1). This upturn represents a radical reversal of the downward trend in the low birthweight rate that had existed since 1968. Between 1968 and 1984 the average monthly percentage of low-birthweight births among Blacks in New York City fell from 13.9 to 10.1; by 1988, the average monthly rate had risen to 12.9. Given the present rate of increase, the rate of low birthweight among Blacks in 1990 will exceed the rate that existed 22 years earlier. This paper applies several statistical methods to the 21 years of monthly, race-specific data from New York City to measure the trend reversal in the rate of low birthweight since 1984. It also estimates the financial costs of such an increase in the incidence of low birthweight.

Methods

Data

The data on low birthweight are from micro-level vital statistics for New York City. Each year of data has been aggregated by month for Blacks and Whites separately from January 1968 through December 1988 (N=252). The data for Whites contain a large component which is Hispanic because ethnicity was not identified on New York City birth certificates until 1978. Moreover, the proportion of births to White women of Hispanic origin has increased over time. In 1978, for instance, 28 percent of all births in New York City were to women of Hispanic descent and 34 percent of all births were to White non-Hispanic women. By 1987, the Hispanic portion had risen to 30 percent while the White non-Hispanic portion had fallen to 29 percent. The implications of a shift in the ethnic distribution of births among Whites are discussed below.

New York City averaged approximately 100,000 resident live births per annum over the time span under study. The percentage of births to Whites and Blacks has varied between 69 percent White and 29 percent Black in 1970 to 57 percent White and 36 percent Black in 1987. Between 1968 and 1988 there were approximately 6,000 White births and 3,000 Black births per month.

Statistical Methods

Two approaches are used to illustrate the changes that occurred in the incidence of low birthweight in 1984. First, the race-specific rate of low birthweight is regressed on a constant, a linear trend term, a dummy variable that equals zero prior to July 1984 and one thereafter, and an interaction trend term. The interaction term is the product of the linear trend term and the dummy variable. An additional set of 11 dummies is included to control for seasonality. A negative coefficient on the trend term and a positive coefficient on the interaction term would be evidence that the downward trend in the rate of low birthweight prior to July 1984 had turned upwards after that point.[2] The sensitivity of the regression estimates are tested by re-estimating the model with January 1984 and January 1985 as the point at which the trend is assumed to have changed.

Another means of illustrating how the low birthweight series have changed involves the use of Box-Jenkins techniques.[3,4] Specifically, an autoregressive-integrated-moving-average model (ARIMA) is fit to each of the series from January 1968 through July 1984. The fitted model is used to project the rate of low birthweight that would have been observed if the underlying process which generated the observed data between 1968 and 1984 had not been altered. The projected rate of low birthweight is compared with the actual rate in order to approximate the additional number of

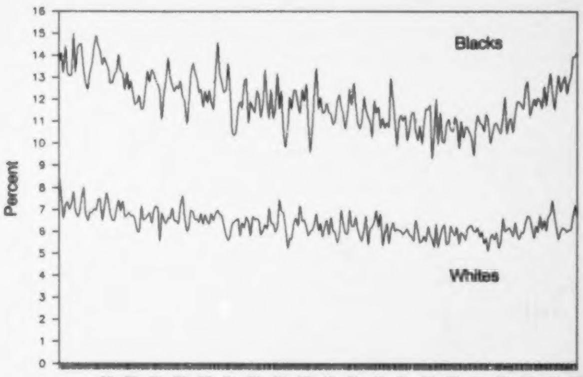


FIGURE 1—Percent of Low-Birthweight Births among Blacks and Whites, New York City, 1968–88

Address reprint requests to Theodore Joyce, PhD, Research Associate, National Bureau of Economic Research, Inc., 269 Mercer Street, 8th Floor, New York, NY 10003. Dr. Joyce is also Associate Professor, Baruch College, City University of New York. This paper, submitted to the Journal November 16, 1989, was revised and accepted for publication February 22, 1990.

Editor's Note: See also related editorial p 655 and article p 679 this issue.

low-birthweight births that have occurred as a result of the shift in the trend. The additional number of low-birthweight births are multiplied by national estimates on the short-term and long-term costs of low birthweight in order to estimate the financial burden of the increase in low birthweight.

Results

Estimates from the linear regression are presented in Table 1. For Whites and Blacks the downward trend in the rate of low birthweight became positive after July 1984 as evidenced by the negative coefficient on the trend term and the positive coefficient on the interaction trend term. The results are insensitive to the switching point of the interaction term from July 1984 to either January 1984 or January 1985 (additional regressions not shown).[*]

We fit ARIMA models to the low birthweight series based on data from January 1968 through June 1984. To eliminate seasonality, a twelfth-order difference was applied to each series; in the case of Blacks, a first-order difference transformation was also needed to achieve stationarity. Based on the autocorrelation and the partial autocorrelation functions, the low-birthweight series for Blacks can be characterized as a second-order moving average with a first-order seasonal moving average. The series for Whites can be characterized by a first-order seasonal moving average. Estimates of the ARIMA models are presented in Table 2. The Ljung-Box Q-statistic supports the adequacy of the model.

The estimated ARIMA models were used to predict the rate of low birthweight for each race from July 1984 through December 1988. A comparison of the actual rates and the projected rates is presented in Figure 2. What is particularly striking is the growing divergence between the actual and the projected low-birthweight rate among Blacks.

To underscore the magnitude of the change, we multiplied the projected rates of low birthweight from July 1984 through December 1988 by the actual number of live births over the same period. The product yields the number of low-birthweight births that would have been expected based on a 54-month forecast made in July 1984. The difference between the actual number of low-birthweight births and the projected number represents an estimate of the excess number of low-birthweight births above what would have been observed had the rate of low birthweight continued a realization consistent with the previous 16 years. For the entire 54-month period beginning in July 1984, we estimate that there have been 3,110 additional Black low-birthweight births and 1,385 White low-birthweight births.

TABLE 1—Estimated Piecewise Linear Regressions of the Rate of Low Birthweight on a Linear Trend Term: Blacks and Whites, 1968,1–1988,12[a]

	Coefficient	Standard Error	P-value
Blacks			
Constant	13.689	.157	.0001
Linear trend	−.016	.001	.0001
Dummy (1 if T > = July 1984)	−14.652	1.718	.0001
Trend × Dummy	.072	.008	.0001
Adjusted R^2	.70		
Whites			
Constant	7.165	.075	.0001
Linear trend	−.007	.001	.0001
Dummy (1 if T > = July 1984)	−3.965	.770	.0001
Trend × Dummy	.020	.003	.0001
Adjusted R^2	.52		

[a] The seasonal dummy variables are not shown. The estimates have been corrected for first-order autocorrelation by means of the Corchrane-Orcutt procedure.[2]

To assess the financial burden of the additional low-birthweight births, we used cost estimates from the US Congress, Office of Technology Assessment.[5] The figures represent the incremental costs of a low-birthweight birth; that is, the costs above those associated with a normal-weight delivery. The costs are broken down into three categories: initial hospitalization, rehospitalization, and long-term treatments. To simplify matters, we grouped initial hospitalization and rehospitalization under the rubric of short-term costs. Long-term costs refer to expenses beyond the first year of life. The report found that, on average, the short-term costs of a low-birthweight birth ranged from a low of $5,040 to a high of $7,525 in 1986 dollars. If long-term costs were added, the total incremental costs varied from $14,040 to $30,525 per low-birthweight birth.

We focus on the short-term costs since these expenses have already been incurred. For Blacks, the total short-term costs associated with the excess number of low-birthweight births range from a low of $15,674,400 to a high of $23,402,750 in 1986 dollars. For Whites, the comparable costs are between $6,980,400 and $10,422,125.

Discussion

The increase in substance abuse is the most likely explanation for the increase in the incidence of low birthweight. New York City birth certificates reveal that between 1981 and 1987 the number of infants born to drug-abusing mothers rose from 6.7 per 1000 live births to 20.3 per 1000 live births, a tripling of the rate.[**] The extent of the underreporting is potentially serious, but the point is unlikely to change: the number of infants exposed to drugs has increased precipitously over the last four years. Moreover, there is a growing body of epidemiological evidence which links the use of cocaine during pregnancy to an elevated risk of low birthweight.[6–8] Finally, no other risk factors reported on birth certificates appear to have undergone sufficient change to account for the increased incidence of low birthweight. Annual data show little variation in the percentage of births to women with late or no prenatal care, the percentage of births born out-of-wedlock, and the percentage of births to women on Medicaid.[1]

The increase in the incidence of low birthweight has had relatively little impact on the neonatal mortality rate. Among Black non-Hispanics, for example, the neonatal mortality rate stood at 13.6 in 1983, 11.8 in 1984, 13.6 in 1985, 12.3 is 1986, and 13.4 in 1987.[1] The provisional figure for 1988 is 14.6.[***] Among White non-Hispanics, Puerto Ricans, and other Hispanics, the neonatal mortality rate has either fallen slightly or remained essentially unchanged since 1983. The mortality rates imply that many of the additional low-

---

[*] A more detailed discussion of the regression results is available upon request to author.

[**] Habel L, Lee J, Kaye K: Trends in Maternal Drug Abuse during Pregnancy in New York City 1978–87. Presented at the 116th Annual Meeting of the American Public Health Association, Boston, MA, Nov. 13–17, 1988.

[***] New York City Health Department, Department of Biostatistics.

TABLE 2—Estimated ARIMA Equations for the Rate of Low Birthweight Blacks and Whites 1969,2–1984,6

Series	General Form (p,d,q) × (P,D,Q)	constant	Parameters ϕ_1	ϕ_2	Φ_{12}	Q-statistic (df = 39)
Blacks	(0,1,2) × (0,1,1)		−.708 (.072)	−.247 (.072)	−.688 (.059)	33.65
Whites	(0,0,0) × (0,1,1)	−.088 (.010)			−.745 (.052)	43.58

* Standard errors are in parentheses. The probability value for the Q-statistic is .71 for Blacks and .23 for Whites.

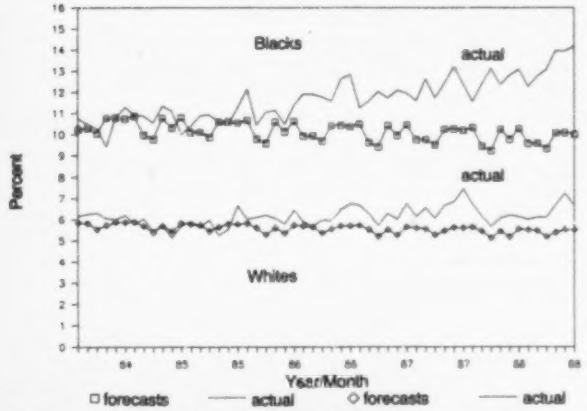


FIGURE 2—Percent of Low-Birthweight Births (forecast vs actual) among Blacks and Whites, New York City, July 1984–December 1988

birthweight infants survive the first month of life. However, the upturn among Blacks in 1988 suggests that the capability of the tertiary care system to manage the increase in low-birthweight births may be limited.

As mentioned above, the proportion of White births to women of Hispanic origin has risen over time. If the rate of low birthweight among Hispanics is higher than that of White non-Hispanic women, then the increase in the incidence of low birthweight among White women may simply reflect the change in the ethnic composition of White births. However, the increase in the proportion of White births to women of Hispanic descent has been dominated by an increase in non-Puerto Rican Hispanics. The incidence of low birthweight and neonatal mortality among this group of Hispanics is almost identical to that of White non-Hispanics.[1] Thus, the growth in the proportion of White births to women of Hispanic origin is not a good explanation for the increased incidence of low birthweight.

The lack of monthly data on substance abuse by pregnant women prevented a more direct test of whether the rise in the proportion of low-birthweight births is associated with the growth in substance abuse. Nevertheless, the increase in the aggregate rate of low birthweight in a city the size of New York is an important, yet troubling finding. At the very least it indicates a deterioration in the health of newborns in New York City. Moreover, anecdotal evidence from the daily media suggests that the use of cocaine, or its derivative crack, among pregnant women is a worsening problem in cities across the United States. If substance abuse is the causal factor behind the unprecedented rise in the incidence of low birthweight in New York City, then one can expect similar increases in low birthweight in other parts of the country. Given the costs of treating low-birthweight children, action taken now may be highly cost-effective.

ACKNOWLEDGMENTS

The research was supported by Grant Number 88-5567 from the Henry J. Kaiser Family Foundation to the National Bureau of Economic Research (NBER). The research is part of the NBER's Program in Health Economics. Any opinions expressed are those of the author and should not assume to be those of the Kaiser Family Foundation or the NBER. I wish to thank Michael Grossman, Mark Berenson, and Nancy Aries for their helpful comments.

REFERENCES

1. New York City Department of Health: Summary of Vital Statistics 1987. New York, NY: Bureau of Health Statistics and Analysis, 1989.
2. Gujarati D: Basic Econometrics, 2nd Ed. New York: McGraw-Hill, 1988.
3. Box GEP, Jenkins GM: Time-Series Analysis: Forecasting and Control, Rev Ed. San Francisco: Holden-Day, Inc, 1976.
4. Vandaele W: Applied Time-Series and Box-Jenkins Models. Orlando: Academic Press, 1983.
5. US Congress, Office of Technology Assessment: Healthy Children: Investing in the Future, OTA-H-345. Washington, DC: Govt Printing Office, 1988.
6. Zuckerman B, *et al:* Effects of maternal marijuana and cocaine use on fetal growth. N Engl J Med 1989; 320:762–768.
7. Kaye K, Elkind, L, Goldberg D, Tutun A: Birth outcomes for infants of drug abusing mothers. N Y State J Med 1989; 89:256–261.
8. Chasnoff IJ, Burns WJ, Schnoll SH, Burns KA: Cocaine use in pregnancy. N Engl J Med 1985; 313:665–669.

Prospective authors should consult "Information for Authors" which appeared in the January issue of this Journal and may be also obtained from the Journal Editorial Office:
American Journal of Public Health
1015 15th Street, NW
Washington, DC 20005

4T

Secular Change in Birthweight among Southeast Asian Immigrants to the United States

De-Kun Li, MD, MPH, Hanyu Ni, MD, Stephen M. Schwartz, MPH, and Janet R. Daling, PhD

Abstract: We analyzed the Washington State births in 1980–1986 to Southeast Asian parents whose birth place was coded as being outside of the US. The mean birthweight increased from 3188 grams in 1980–81 to 3283 grams in 1986, an annual increment of about 18 grams (95% CI = 11, 25 grams). The prevalence of low birthweight (<2500 grams) decreased from 7.2 percent in 1980–81 to 5.4 percent in 1986, an annual reduction of 6.4 percent (95% CI = 1.3, 11.2 percent). In an analysis of information ascertained from the birth certificate, the change of paternal occupational status (from student to employed) was associated with 27 percent of the reduction in the prevalence of low birthweight, independent of maternal age, infant sex, and prior gravidity. A similar temporal change of birthweight during the same period of time was not observed among infants of US-born Asian mothers. Among the women who had two consecutive births from 1984 to 1986, the improvement of birthweight for the second birth compared to the first birth was much greater in infants born to the Southeast Asian mothers than that to White mothers. Our study suggests that living in the United States after immigration from their native countries has had a positive impact on the birthweight of infants born to Southeast Asian immigrants. (*Am J Public Health* 1990; 80:685–688.)

Introduction

Since the mid 1970s, approximately 800,000 Southeast Asian refugees have immigrated to the United States.[1] At the time of their arrival, the majority of these refugees were of lower socioeconomic status than the general population of the US, and had undergone severe hardships.[2] Health assessment by local and federal agencies revealed several serious problems among this population, including nutritional deprivation, anemia, and psychiatric disorders.[3–7] In addition, it has been reported that they lacked adequate prenatal care and were at high obstetric risk.[8] Since their arrival, however, several programs have been established to help these recent immigrants with financial support, education, and health care.[2] This study attempted to evaluate whether or not the risk of low birthweight has changed over time among Southeast Asian immigrants and, if so, to determine what factors may have accounted for any change observed.

Methods

Washington State contains one of the largest populations of Southeast Asian refugees[2]; the peak wave of the immigrants occurred in the late 1970s and early 1980s.* All births between 1980 and 1986 to Washington State residents (both mother and father) whose race was coded as "other Asian" (i.e., Asian but not Chinese, Japanese, or Filipino) and for whom place of birth was coded as "outside US" were selected from computerized files of Washington State Birth Certificates. We reviewed the original certificates of a random 5 percent sample of the selected births for each year to estimate the proportion of those births to parents who were truly born in Southeast Asia. About 70 percent were born to parents from Southeast Asian countries, primarily Vietnam, Laos, and Cambodia, while the majority of the remainder were born to parents from Korea, India, and the Pacific Islands. The following information on each birth available from the file of birth certificates was used in this study: birthweight, maternal age, marital status, gravidity, parity, sex of infant, the number of prenatal visits, the month of first prenatal visit during pregnancy, method of delivery, pregnancy and labor complications, malformation status, mother's smoking status during the pregnancy (ever vs never, available only for the years 1984–86), number of prior spontaneous and induced abortions, and mother's and father's occupations.

The infants born in 1980 and 1981 were taken as the reference cohort that presumably reflected the situation shortly after the immigrants initially arrived in the United States. Births in 1982 to 1986 among this population thus comprised successive comparison cohorts, assuming that the parents of these infants were those that had been in the United States for progressively greater lengths of time. To evaluate whether or not any changes of birthweight in the study population were part of a general trend among births to all Asian parents in Washington State, we also analyzed the 770 births available during the study period to Washington residents (both parents) whose race was indicated as being a specific Asian ethnic group (i.e., Chinese, Japanese, Filipino) or other Asian, and for whom the place of birth was coded as any one of the 50 US States or the District of Columbia.

Average annual changes in the proportion of low birthweight (<2500 grams) and high birthweight (≥ 3500) were estimated using logistic regression for dichotomous outcomes,[9] while corresponding changes in mean birthweight were estimated using multiple linear regression for continuous outcome.[10] The odds ratio was used as an approximation of the relative risk when dichotomous outcomes of birthweight were evaluated, i.e., the proportion of low birthweight and high birthweight. Although the proportion of the birthweight equal to or greater than 3500 grams is not a rare event (it occurred in about 20 to 30 percent of births in our study), the discrepancy between the odds ratio and relative risk for this outcome is practically negligible. To estimate the proportion of annual change in birthweight accounted for by specific demographic and obstetric factors, we determined the difference between the estimates of annual change

---

Address reprint requests to Janet R. Daling, PhD, Fred Hurchinson Cancer Research Center (MP381), 1124 Columbia Street, Seattle, WA 98104. She is also Professor, Department of Epidemiology, School of Public Health and Community Medicine, University of Washington. The co-authors are Research Associates at the Fred Hutchinson Cancer Research Center. This paper, submitted to the Journal January 6, 1989, was revised and accepted for publication November 15, 1989.

*Department of Public Health, Refugee Health Service of Seattle-King County: Two-thirds of the Southeast Asian immigrants to Washington State reside in Seattle-King County.

adjusted and unadjusted for the specific factors and divided this figure by the unadjusted estimates of annual change.

To examine more explicitly whether or not increased duration of residence in the US has influenced the distribution of birthweight among Southeast Asian immigrants, data were also analyzed on those Washington State parents who gave birth to two consecutive children during years 1984–87, and who met the same race and birthplace criteria as described above (data from 1980–83 were not available). Thus, the birthweight of the second child born during this period (not necessarily the second child ever born to each woman) was compared to that of preceding birth. In order to distinguish any secular change of birthweight among Southeast Asian immigrants from that among general population in the Washington State, we also analyzed White women who gave birth to two consecutive children during the period of 1984–87 (there were too few US-born Asian women for similar analyses of this type). The risks of low birthweight for Southeast Asian immigrants compared to American Whites were then calculated separately for each of the two births occurring during the years 1984–87. The change in low birthweight among Southeast Asian immigrants beyond the secular change in the general population in the State was computed by dividing the difference of odds ratio between two consecutive births by the odds ratio minus one for the first birth:

$$\frac{(OR_{\text{second birth}}) - (OR_{\text{first birth}})}{(OR_{\text{first birth}}) - 1}$$

Results

The prevalence of low birthweight (<2500 grams) decreased among Southeast Asian immigrants from 7.2 percent in 1980–81 to 5.4 percent in 1986, a reduction of almost 25 percent (RR = 0.75, 95% CI = 0.53, 1.03) (Table 1). During this same time period, the proportion of birthweight equal to or greater than 3500 grams increased from about 24 percent in 1980–81 to 30 percent in 1986, a 26 percent increment (RR = 1.26, 95% CI = 1.09, 1.45). The mean birthweight increased steadily from 3188 grams in 1980–81 to 3283 grams in 1986 with an average increase of 95 grams (95% CI = 62, 120 grams) during the six-year period (Table 1). A similar secular change of birthweight distribution was not observed among US-born Asian population (data available on request to authors).

The analyses of the data from those subjects with two consecutive births during 1984–87 revealed that the prevalence of low birthweight among Southeast Asian immigrants declined 55 percent (OR = 0.45, 95% CI = 0.23, 0.86) in the second birth compared to the preceding birth. Compared to Whites, there was a 92 percent [(1.05-1.61)/(1.61-1)] reduction, after adjustment for maternal age and parity, in the risk of low birthweight for the second birth compared to the preceding birth among Southeast Asian immigrants beyond the secular change among general population (Table 2).

TABLE 1—Distribution of Birthweight (BW) among Immigrants from Southeast Asia, by Time Period, Washington State, 1980–86

Time Period (N)	BW < 2500 gms % (n)	BW ≥ 3500 gms % (n)	Mean BW grams (SE)
1980–81 (2094)	7.2 (150)	23.8 (498)	3188 (11)
1982 (1210)	7.6 (92)	25.8 (312)	3190 (15)
1983 (976)	5.8 (57)	29.2 (285)	3238 (16)
1984 (1008)	5.8 (58)	27.1 (273)	3241 (16)
1985 (1062)	5.9 (63)	28.0 (297)	3243 (15)
1986 (994)	5.4 (54)	30.0 (298)	3283 (17)

Based on data available from the birth certificate (see Methods), we compared the temporal changes in factors considered *a priori* to be associated with birthweight to the changes of birthweight during the same period of time (Table 3). There were large increases during the study period in the proportion of women giving birth after age 30 and in the primary cesarean section rate. There was also a 60 percent decrease in the proportion of fathers who listed "student" as their occupation. Aspects of reproductive history, maternal marital status, level of prenatal care, and prevalence of cigarette smoking during pregnancy showed little or no variation between 1980–81 and 1986 although information on smoking during pregnancy was only available for the last three years.

From 1980–81 to 1986, there was a 6.4 percent annual reduction in the prevalence of low birthweight, a 5.8 percent annual increase in the proportion of high birthweight (≥3500 grams), and a 17.7 gram annual increment of mean birthweight among immigrants from Southeast Asia (Table 4). Temporal variation of maternal age, infant sex ratio, and prior gravidity explained between 17 and 28 percent of the secular change in birthweight (depending upon birthweight measure) and, beyond the explanatory power of these trends, the change in paternal student status accounted for an additional 16 to 27 percent of the change in birthweight. Together, maternal age, infant sex, gravidity, and paternal occupational status accounted for between 42 and 55 percent of the change in birthweight among births to immigrants from Southeast Asia, and inclusion of other aspects of obstetrical history, prenatal care, method of delivery, infant malformation status, and demographic characteristics in the models did not confound the above results or explain the remaining unexplained portion of the change in birthweight parameters.

Discussion

There are several limitations in our analyses. Most important, we did not review the more than 7,000 birth certificates to distinguish births to Southeast Asian immigrants from births to parents of other ethnic groups also categorized by Washington State as "other Asian." Our review of a random sample of these certificates indicates that if such a distinction were possible, about 70 percent of the study population would be classified as Southeast Asian in origin. However, our review of the birth certificates also

TABLE 2—Comparison of Low Birthweight (LBW) between two Consecutive Births among Southeast Asian Immigrants and American Whites, Washington State, 1984–87

	Proportion of LBW[a] (N)			
Birth Sequence	SE Asian % (N)	White % (N)	OR[b]	95% CI
First[c]	6.6 (423)	4.3 (21562)	1.61	1.1, 2.4
Second[d]	3.1 (425)	2.9 (21570)	1.05	0.6, 1.8

[a] <2500 grams.
[b] Adjusted for maternal age and parity.
[c] Three Southeast Asians and 69 Whites with missing data.
[d] One Southeast Asian and 61 Whites with missing data.

TABLE 3—Distribution (%) of Characteristics among Immigrants from Southeast Asia, by Time Period, Washington State, 1980–86

Factors	1980–81 (N = 2105)[a,b]	1982 (N = 1214)	1983 (N = 976)	1984 (N = 1015)	1985 (N = 1064)	1986 (N = 998)
Maternal age (years)						
<20	6.6 (139)	6.1 (74)	5.7 (56)	3.4 (35)	4.0 (43)	2.8 (28)
20–24	27.8 (584)	31.9 (386)	24.2 (236)	23.5 (239)	19.7 (210)	19.8 (197)
25–29	36.4 (764)	35.3 (428)	38.4 (375)	37.5 (381)	39.7 (422)	37.0 (369)
≥30	29.2 (614)	26.7 (323)	31.7 (309)	35.5 (360)	36.6 (389)	40.4 (403)
Infant sex						
Male	50.7 (1067)	50.5 (613)	54.2 (529)	49.1 (498)	52.5 (559)	53.4 (553)
Female	49.3 (1038)	49.5 (601)	45.8 (447)	50.9 (517)	47.5 (505)	46.6 (465)
Prior gravidity						
0	28.6 (589)	28.5 (342)	27.5 (263)	30.1 (295)	27.5 (282)	26.8 (259)
1–2	46.0 (948)	49.0 (589)	46.9 (448)	46.2 (452)	47.5 (488)	49.2 (476)
≥3	25.4 (524)	22.5 (270)	25.6 (245)	23.7 (232)	25.0 (257)	24.0 (232)
Primary cesarean section						
Yes	6.0 (127)	6.2 (75)	8.1 (79)	7.3 (74)	8.0 (85)	10.9 (109)
No	94.0 (1976)	93.8 (1139)	91.9 (897)	92.7 (941)	92.0 (979)	89.1 (889)
Paternal occupation						
Student	27.7 (563)	24.2 (290)	15.1 (144)	11.4 (114)	10.0 (103)	10.1 (100)
Other	72.7 (1471)	75.8 (910)	84.9 (811)	88.6 (882)	90.0 (922)	89.9 (887)

[a] Numbers in parentheses are number of live births.
[b] The number of subjects for an individual factor may not match the total number of subjects due to missing data.

TABLE 4—Annual Change in Birthweight among Immigrants from Southeast Asian, Washington State, 1980–86

Birthweight Measures	Crude	Adjustment 1[c]	Adjustment 2[d]
Percent change in the prevalence of low birthweight (<2500 grams)[a]	−6.4	−4.6 (28.1)[e]	−2.9 (54.7)
(95% CI)	(−1.3, −11.2)	(0, −9.6)	(+2.6, −8.1)
Percent change in proportion of high birthweight (≥3500 grams)[a]	5.8	4.8 (17.2)	3.4 (41.4)
(95% CI)	(2.8, 8.8)	(1.8, 8.0)	(0.3, 6.5)
Change in mean birthweight (grams)[b]	17.7	13.1 (26.0)	10.3 (41.8)
(95% CI)	(10.8, 24.6)	(6.2, 20.0)	(3.4, 17.2)

[a] Estimated from logistic regression.
[b] Estimated from multiple linear regression.
[c] Estimates following adjustment for maternal age, infant sex, and prior gravidity categorized as shown in Table 3.
[d] Estimates following adjustment for paternal student status in addition to adjustment for maternal age, infant sex, and prior gravidity.
[e] The numbers in parentheses are the proportion of crude estimates that can be explained by the adjusted factors.

indicates that the proportion of parents who were born in Southeast Asia did not change appreciably over the study period. Since there is little reason to expect that Asians not born in Southeast Asia who were included in our study population would have experienced any noticeable change in their birthweight distribution, the inclusion of such misclassified births (with respect to race) would only lead to an attenuation of the true trends among births to Southeast Asian immigrants.

Another possible source of misclassification that may have arisen in our study is that calendar year (the independent variable in our analyses) is not a perfect correlate of length of time of residence in the US, since some immigrants from Southeast Asia continued to arrive in Washington State from abroad each year, and some immigrated to the US before 1980. Thus, a proportion of the births to Southeast Asian immigrant parents in later years of the study period might have been to recent immigrants, while a proportion of the births in the earlier years of the study period might have been to parents who had been in the US since the mid-1970s. If the health status of more recent immigrants is better than that of past immigrants, which might be reflected in an improved birthweight outcome of their offsprings, the attribution of change of distribution of birthweight that we observed to the length of residence in the US may be spurious. However, our analysis of consecutive births to the same parents during the latter half of the study period showed a decline in low birthweight among immigrants from Southeast Asia which can not be attributable to misclassification of recency of migration. In addition, there is evidence that the health status of recent Southeast Asian immigrants has not improved (at least compared to earlier immigrants) as measured by the prevalence of hepatitis B surface antigen carriers and TB infection.** We therefore believe that this source of misclassification would also lead to observed trends which are, if anything, diminished in magnitude compared to the actual secular changes.

Information on a major risk factor for low birthweight, maternal cigarette smoking during pregnancy, was only available for the three most recent years of the study period. Nevertheless, the proportion of births to mothers who smoked during pregnancy was very low in these years, and was relatively stable, ranging from 3 percent in 1984 to 2.4 percent in 1986. Thus, we feel that our inability to account for changes in cigarette smoking status over the entire study period is unlikely to have explained our results.

The majority of immigrants from Southeast Asia during this period came from underdeveloped parts of their native countries.[1–3] Some of them had experienced nutritional deprivation, starvation, disease infection, and horrible tortures prior to their arrival in the United States.[2–5] Immigration to the United States and the subsequent improvement of their economic and health status might be expected to have

---

**Cochran J: Director of Refugee Health Program, Department of Public Health, Massachusetts. (personal communication) some impact on pregnancy outcomes in this population, particularly birthweight. Our study, conducted over a seven-year period, provides evidence that living in the United States has improved the birthweight distribution of infants born to immigrants from Southeast Asia since the time of initial immigration. Of the information available on the birth certificate, secular changes in maternal age, male sex, prior gravidity, and paternal occupation were all correlated with the increase in birthweight. Of these factors, the change in father's occupational status (from "student" to "employed") is the one which is most likely to reflect the change of the Southeast Asian immigrants' socioeconomic environment, which may include improved nutrition, family security, and better adaptation to a new culture. Further research is needed to identify specific factor(s) that might have contributed to the change of birthweight distribution among this population.

ACKNOWLEDGMENTS

The authors thank the Department of Social and Health Services, Health Data Section, of Washington State for allowing the use of birth certificate data, and John T. Riess of the Department of Public Health, Refugee Health Service of Seattle-King County for providing the information on the number of new arrivals of Southeast Asian immigrants to Seattle-King County by year. This study was presented at the 116th Annual Meeting of American Public Health Association in Boston, Massachusetts, November 14–17, 1988.

REFERENCES

1. Refugee Program. Monthly summary, September 1986. Washington, DC: Department of State, September 30, 1986.
2. Marsh RE: Socioeconomic status of Indo-Chinese refugees in the United States: Progress and problems. Social Security Bull 1980; 43:10.
3. Erickson RV, Hoang GN: Health problems among Indo-Chinese refugees. Am J Public Health 1980; 70:1003–1006.
4. Wiesebtgak AM, Nickele MK, Hashimoto G, Endo T, Ehrhard H-B. Intestinal parasites in Southeast Asian refugees—prevalence in a community of Laotians. JAMA 1980; 244:2543–2544.
5. Waldman EB, Lege B, Oseid B, *et al:* Health and nutritional status of Vietnamese refugees. So Med J 1979; 72:1300–1303.
6. Aduan RP, McCutcheon M: Refugees from Indochina. Ann Intern Med 1980; 92:266.
7. Health Status of Indo-Chinese Refugees. MMWR 1979; 28:385–389.
8. Davis JM, Goldenring J, McChesney M, Medina A: Pregnancy outcomes of Indo-Chinese refugees, Santa Clara County, California. Am J Public Health 1982; 72:742–744.
9. Breslow NE, Day NE: Statistic Methods in Cancer Research Vol 1—analysis of case-control studies. Lyon: IARC Sci. Pub. 1980; 14–247.
10. Kleinbaum DG, Kupper LL: Applied Regression Analysis and Other Multivariable Methods. Boston: Duxbury Press, 1978.
11. Taffel S: Factors associated with low birthweight. Pub. (PHS) 1980: 1915. National Center for Health Statistics, 1980; Series 21, No. 37.
12. Meredoth HV: Body size of infants and children around the world in relation to socioeconomic status. Adv Child Dev Behav 1984; 18:81–145.

NIH Consensus Development Conference on Early Stage Breast Cancer

Treatment of early stage breast cancer is the subject of an upcoming Consensus Development Conference sponsored by the National Institutes of Health (NIH). The conference, scheduled for June 18–21, 1990 in Masur Auditorium of the NIH Clinical Center, is open to the public.

The purpose of the conference is to reach agreement on the treatment of early stage breast cancer. Key issues to be addressed are:

- The roles of mastectomy versus breast conservation,
- Optimal techniques for breast conservation,
- Adjuvant therapy for patients with node negative breast cancer,
- Use of prognostic factors in managing node negative breast cancer,
- Directions for future research.

The conference will bring together specialists in oncology, surgery, clinical trials, and other relevant fields to present current scientific thinking about the diagnosis, management, and prevention of breast cancer. William C. Wood, MD, Chief of Surgical Oncology, Massachusetts General Hospital, will chair the panel, which will produce the draft report and invite comments from the conference participants. To register for the conference or to obtain further information, contact:

Conference Registrar
Prospect Associates
Suite 500
1801 Rockville Pike
Rockville, MD 20852
Tel: 301/468-meet

2T

Potential Exposure and Health Risks of Infants following Indoor Residential Pesticide Applications

RICHARD A. FENSKE, PHD, MPH, KATHLEEN G. BLACK, MPH, KENNETH P. ELKNER, MS, CHORNG-LI LEE, MS, MARK M. METHNER, MS, AND RALPH SOTO, MS

Abstract: Air and surface chlorpyrifos residues were measured for 24 hours following a 0.5 percent Dursban® broadcast application for fleas inside a residence. Two of the three treated rooms were ventilated following application. Maximum air concentrations were measured 3–7 hours post-application. Peak concentrations in the infant breathing zone were 94 $\mu g/m^3$ in the nonventilated room and 61 $\mu g/m^3$ in the ventilated room, and were substantially higher than concentrations in the sitting adult breathing zone. Concentrations of approximately 30 $\mu g/m^3$ were detected in the infant breathing zone 24 hours post-application. Surface residues available through wipe sampling were 0.7–1.6 $\mu g/cm^2$ of carpet on the day of application and 0.3–0.5 $\mu g/cm^2$ 24 hours post-application. Estimated total absorbed doses for infants were 0.08–0.16 mg/kg on the day of application and 0.04–0.06 mg/kg the day following application, with dermal absorption representing approximately 68 percent of the totals. These doses are 1.2–5.2 times the human No Observable Effect Level (NOEL). Exposures to cholinesterase inhibiting compounds following properly conducted broadcast applications could result in doses at or above the threshold of toxicological response in infants, and should be minimized through appropriate regulatory policy and public education. (*Am J Public Health* 1990; 80:689–693.)

Introduction

The health risks associated with residual pesticides in air and on surfaces in residences have only recently been examined from a public health perspective. The Committee on Urban Pest Management established by the US National Academy of Sciences noted that 5,000 health-related incidents involving pesticides were reported as occurring in homes in the US from 1966 to 1979.[1] Home pesticide applications may pose health risks for occupants, particularly infants and children. A study of 20 adult residents exposed to dichlorvos following crack-and-crevice treatments found a slight decrease in group mean serum cholinesterase, with three residents reporting a feeling of illness 24 hours post-application.[2] Six of 21 children admitted to Arkansas Children's Hospital for organophosphate intoxication were judged to have been exposed following insecticide spraying inside the home.[3] A review of 37 hospitalized pesticide poisonings among infants and children at the Children's Medical Center in Dallas, Texas revealed that five cases were due to pesticide absorption following spraying or fogging inside residences.[4] California State agencies reviewed 44 illness reports in 1984 associated with residential pesticide use,[5] and subsequently conducted a hazard assessment for acute intoxication potential among infants following indoor pesticide treatments.[6] Concern also exists regarding the chronic health effects of children's exposure to pesticides.[7]

Residential monitoring studies have focused primarily on ambient air concentrations of pesticides following several types of applications, including termiticide applications,[8–13] DDVP resin strips hung in households,[14–16] and crack-and-crevice or baseboard treatments.[17–22] Recently the US Environmental Protection Agency initiated a major study of pesticide air concentrations in homes.[23] Limited studies of two additional application methods have been conducted. Broadcast applications,[24,25] involve the hand spraying of an aqueous mixture directly onto target surfaces (e.g., rugs, floors, furniture), while total aerosol release applications (foggers)[26–28] involve activation of a pressurized canister in an unoccupied room, with the pesticide aerosol covering all surfaces (walls, floor, ceiling). Both of these methods produce residues on surfaces to be contacted by occupants. Flea treatment by broadcast application was selected for study here because of the potential for both high air concentrations and substantial dermal exposure. A preliminary risk assessment has been conducted for crawling infants (9–10 months of age).

Methods

Sampling was conducted according to guidelines developed by the Canadian government's Department of Health and Welfare.[29] The study was conducted in three rooms of an unoccupied apartment in New Jersey in June 1987, and extended for 24 hours following application. All rooms were carpeted wall-to-wall with 1.2 cm thick nylon pile. Total surface area treated was 45.27 m^2. No heating or air conditioning systems were in operation during the study. Windows or doors were open in two of the three rooms, while no ventilation occurred in the third room.

Chlorpyrifos application for flea treatment was conducted by a licensed pest control applicator. The formulated product, Dursban® L.O. (EPA Registration No. 464-571), contains 41.5 percent chlorpyrifos (O,O-diethyl O-(3,5,6-trichloro-2-pyridyl) phosphorothioate). The formulation was applied approximately 40 cm above the carpet as a 0.50 percent aqueous spray (40 ml/3.785 L water) with a hand-held fan broadcast nozzle attached to a CO_2 pressurized tank. Applications in the three rooms were identical, covering the entire floor and lasting 5–7 minutes per room.

Air samples were collected in duplicate in each room prior to application and at the following time intervals post-application: 0–0.5 hours, 0.5–1, 1–1.5, 1.5–3, 3–5, 5–7. An additional two-hour sample was collected 24 hours post-application. The samplers were placed in the center of each room mounted 100 cm and 25 cm above the treated surface, approximately 1.5 m apart. Battery-powered porta-

---

Address reprint requests to Richard A. Fenske, PhD, MPH, Assistant Professor, Department of Environmental Sciences and Graduate Program in Public Health, Rutgers University, P.O. Box 231, New Brunswick, NJ 08903. Co-authors Black, Elkner, Lee and Methner are graduate assistants, Graduate Program in Human Exposure Assessment/Environmental Sciences, Rutgers University and Robert Wood Johnson Medical School; Soto is a right-to-know trainer, State University of New York at Brooklyn. This paper, submitted to the Journal February 23, 1989, was revised and accepted for publication October 4, 1989.

ble air sampling pumps with flow rates of 2 L/min were calibrated with the sampling train in line prior to and following the sampling period. Chlorpyrifos vapors were collected with ORBO-44 tubes (Supelpak 20). Tubes were capped and stored in an ice chest for transport to the laboratory.

Aluminum foil squares (10 cm × 10 cm) were placed in the rooms before treatment to measure residues deposited on the carpet surface. Five squares were collected from each room immediately following application (5 minutes). The wipe procedure was based on the standard method of the US Occupational Safety and Health Administration.[30] Sets of three wipes were collected in each room at eight time intervals post-application: 20, 40, 80, 100, 170, 260, 380 minutes, and 24 hours. All areas to be wiped were marked by disposable cardboard frames with 10 cm × 10 cm cut-out interiors. A new plastic glove was worn for each wipe sample to avoid cross-contamination of samples. The wipe consisted of three strokes across the surface with a 7.5 × 7.5 cm surgical gauze pad sprayed lightly with distilled water. This procedure was repeated with a second gauze pad, changing the wipe orientation 90°. Pads were placed in glass storage jars in an ice chest for transport to the laboratory.

All samples were stored at −20°C until analysis. Granular contents of the Orbo-44 tubes were transferred into serum vials filled with 10.0 ml of pesticide-grade toluene. A subset of the samples were analyzed for breakthrough into the secondary section of the tubes, but such breakthrough was negligible. Capped vials were shaken at high speed (100 cpm) for 5 minutes on a mechanical shaking table, allowed to sit for 30 minutes and the extracts transferred to GC vials for analysis. Both the foil and the pads were extracted in 30 ml of toluene on the shaking table at high speed for 30 minutes and the extracts transferred into GC vials.

Samples were analyzed on a Hewlett Packard 5890A gas chromatograph equipped with an electron capture detector. Heptachlor epoxide was added to all samples as an internal standard. The limit of detection for 1-μl injections was 1 picogram/μl. The limits of detection for the sampling media were as follows: air samples, 83 ng/m^3; foil and gauze samples, 0.6 ng/cm^2. Solvent blanks run with each set of samples had no detectable chlorpyrifos. Fortification/recovery studies were conducted for all sampling media. The mean and standard deviation from these studies were as follows: air samples, 85.3% ±6.0; aluminum foil squares, 98.6% ±2.5; gauze wipes, 84.0% ±10.1. Data have been adjusted on the basis of these recovery values. Two air samples, three foil and three wipe samples were collected in each room prior to application. No background chlorpyrifos levels were detected.

Results

Chlorpyrifos levels on aluminum foil samples collected immediately (5 minutes) following application averaged 13.6 μg/cm^2 ($\pm$6.1) and levels in the three rooms were not significantly different. Measured deposition corresponded well with the 13 μg/cm^2 value calculated from label recommendations.

Chlorpyrifos residues available by wipe sampling in the ventilated rooms dropped by more than one-half within 30 minutes, averaging 0.7 μg/cm^2 throughout the remainder of Day 1 and 0.3 μg/cm^2 24 hour post-application (Table 1). Surface residues in the nonventilated room followed the same pattern, but were significantly higher at all time intervals.

Chlorpyrifos vapors measured in the sitting adult breathing zone (100 cm above the carpet) increased over time (Figure 1). While concentrations in the ventilated rooms never exceeded 20 μg/m^3, levels $>$60 μg/m^3 were measured in the nonventilated room 3–5 hours post-application. The effect of ventilation was further reflected in samples 24 hour post-application: $>$40 μg/m^3 with no ventilation compared to $<$10 μg/m^3 with ventilation.

Substantially higher chlorpyrifos concentrations were measured in the infant breathing zone (25 cm above carpet), with ventilation having far less impact (Figure 2). Concentrations reached 94 μg/m^3 5–7 hours post-application in the nonventilated room and exceeded 60 μg/m^3 3–5 hours post-application in the ventilated rooms. After 24 hours an equilibrium concentration in all rooms of approximately 30 μg/m^3 was reached. It thus appears that a vertical concentration gradient developed following the application, with the treated carpet serving as a source of volatilized chlorpyrifos. Open windows provided mixing and dilution of air one meter above the carpet, but had much less effect on concentrations near the floor.

Time-weighted averages for the 24 hour post-application period in the infant breathing zone were 41.2 and 66.8 μg/m^3 for ventilated and nonventilated rooms, respectively, substantially higher than the corresponding values of 12.3 and 45.7 μg/m^3 for the adult breathing zone. All of these concentrations exceeded the National Academy of Sciences (NAS) interim guideline of 10 μg/m^3 for chlorpyrifos in indoor air following termiticide treatments.[31]

A risk assessment was conducted based on the scenario of a 9–10 month old infant playing in a carpeted room beginning at least one hour after a 0.5 percent chlorpyrifos

TABLE 1—Chlorpyrifos Residues Available by Wipe Sampling: Effects of Time and Ventilation

	Ventilated Rooms			Nonventilated Room		
Sample Time (hr)	N	Mean Residue[a] (μg/cm^2)	Percent Initial Deposit†	N	Mean Residue[b] (μg/cm^2)	Percent Initial Deposit
<0.5 Immediate	6	1.69	12.4	3	3.90	28.6
0.5–6.5 Day 1	35*	0.69	5.0	18	1.56	11.4
24 Day 2	6	0.28	2.0	3	0.48	3.5

a,b: all ventilated room values significantly lower than nonventilated room values (ANOVA: Student-Neuman-Keuls Test; $p < .01$)

†deposition following application = 13.6 μg/cm^2

*25.2 μg/cm^2 value excluded as outlier

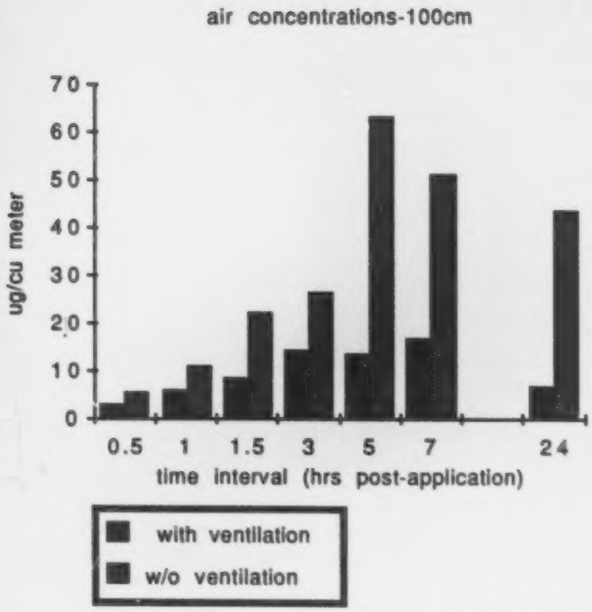


FIGURE 1—Chlorpyrifos Air Concentrations in the Sitting Adult Breathing Zone (100 cm)

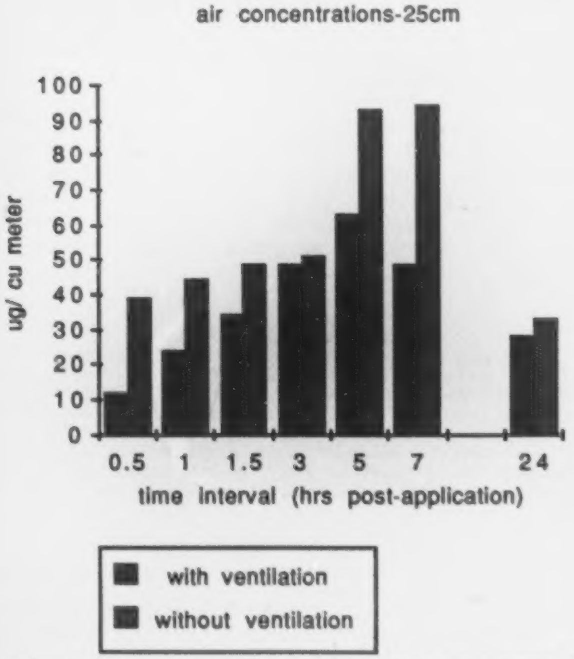


FIGURE 2—Chlorpyrifos Air Concentrations in the Infant Breathing Zone (25 cm)

broadcast application. The following assumptions were employed:

1. Infant weight = 9.0 kg[32]
2. Infant respiratory volume = 2.1 L/min resting (16 hours/day); 6.3 L/min active (8 hours/day)[33]
3. 100% respiratory absorption of chlorpyrifos vapors
4. Total carpet contacted in one day = 2.3 m^2 (25 ft^2)
5. 100% transfer of available residues (wipe samples) to skin
6. 3% dermal absorption of chlorpyrifos[6,34]

Estimated absorbed dermal doses for infants (Table 2) are the product of surface area contacted, available residues (Day 1 and Day 2 in Table 1) and percent absorbed, divided by body weight. The estimated dose in the nonventilated room is 0.120 mg/kg on Day 1, 3.2 times the dose for Day 2. Ventilation reduced the dose estimates approximately twofold. Estimated absorbed respiratory doses are the product of time-weighted air concentrations, respiratory volume and percent absorbed, divided by body weight. Estimated Day 1 dose with no ventilation is 0.038 mg/kg, more than twice the Day 2 dose. Ventilation reduced the dose on Day 1 by approximately one-third, but had little effect on Day 2 estimates. Total absorbed dose estimates for the day of application are 0.075 and 0.158 mg/kg for ventilated and nonventilated rooms, respectively. Day 2 estimates are approximately 2–3 times lower. Dermal exposure represents on average 68 percent of the total absorbed dose.

Discussion

Chlorpyrifos residues following broadcast application were substantially higher than those reported for other types of residential applications. Total release aerosol applications have produced deposition levels of approximately 2 $\mu g/cm^2$ and maximum air concentrations following ventilation of 14 $\mu g/m^3$.[28] Crack-and-crevice or baseboard applications have resulted in deposition levels of from 1–3 $\mu g/cm^2$,[18,22] and air concentrations of 1–2 $\mu g/m^3$.[20,21] Termiticide applications have produced air concentrations of <1–8.5 $\mu g/m^3$.[11-13] Broadcast applications thus appear to produce deposition levels 5–10 times higher and peak air concentrations approximately one to two orders of magnitude higher than other application procedures. Of further concern is the finding that vapor concentrations increase over the first few hours, peaking 3–7 hours post-application.

The extent to which indoor pesticide applications might produce intoxications among residents is largely unknown. A recent risk assessment calculation of a 2.68 mg/kg absorbed dose for infants in chlorpyrifos-treated residences was based on several conservative assumptions, including complete (100 percent) absorption of chlorpyrifos through the skin.[6] The risk assessment presented here avoids such worst-case assumptions, but includes major uncertainties in the dermal exposure estimates. The rate of transfer of pesticides from treated surfaces to skin and the amount of surface contacted over time are not known. Research has been initiated to adapt fluorescent tracer and video imaging techniques used in occupational exposure studies to address these issues.[35,36] Several factors not included in the dose calculations suggest that chlorpyrifos absorption could be substantially higher. First, a 3 percent dermal absorption estimate may be low. The conventional topical dose for pesticide absorption studies is approximately 40 $\mu g/cm^2$,[37] whereas the average dose applied to the skin in the chlorpyrifos absorption study was >4000 $\mu g/cm^2$.[34] High topical doses result in relatively low percent absorbed values.[38,39] An estimate of the rate of chlorpyrifos penetration through the skin is needed to more accurately estimate absorbed dermal dose. Second, infant skin may be more permeable than the adult skin used to estimate chlorpyrifos absorption. Although the stratum cor-

TABLE 2—Total Estimated Absorbed Chlorpyrifos Dose for Infants: Day of Application (Day 1) and Day following Application (Day 2)

Room Condition	Dermal Dose (mg/kg)	% Total	Respiratory Dose (mg/kg)	% Total	Total Dose (mg/kg)	Percent of NOEL*
Day 1						
Ventilated	0.052	69	0.023	31	0.075	250
Nonventilated	0.120	76	0.038	24	0.158	527
Day 2						
Ventilated	0.022	58	0.016	42	0.038	127
Nonventilated	0.037	67	0.018	33	0.055	183

*Human No Observable Effect Level = 0.03 mg/kg/day

neum of the infant is considered to be fully developed, the permeability of infant skin in humans has not been measured.[39] Studies in infant rats have demonstrated increased percutaneous absorption of chlorpyrifos relative to adults.[40] Third, ingestion from deposits on hands would increase the estimates, since chlorpyrifos is absorbed by the oral route more efficiently than by the dermal route.[34] Finally, skin conditions to which infants may be particularly susceptible (e.g., dryness and rashes) are known to reduce the barrier properties of the skin.[39]

The no observable effect level (NOEL) for chlorpyrifos is based on measurable changes in plasma acetylcholinesterase. The NOEL in humans is 0.03 mg/kg/day.[41] All of the absorbed dose estimates in Table 2 exceed the NOEL, with doses 2.5–5 times the NOEL on Day 1, declining to 1.2–1.8 times on Day 2. The US Environmental Protection Agency has derived a "human reference dose" of 0.003 mg/kg/day for chlorpyrifos by applying a 10-fold safety factor to the human NOEL.[42] The dose estimates in this study exceed the human reference dose by 10–50 times.

NOEL values are commonly employed to determine standards for human safety, but are based on an assumption of continuous exposure (e.g., Acceptable Daily Intakes of pesticide residues in foods). A similar assumption underlies the NAS guideline for indoor air concentrations.[30] Since the potential exposures calculated here would be intermittent, their toxicological significance is difficult to interpret. As yet no short-term toxicological measure analogous to the NOEL is available.

Despite uncertainties in exposure/absorption estimates and toxicological interpretation, the dose values derived in this study raise a public health concern. Broadcast applications and possibly total release aerosol/fogging applications of acutely toxic insecticides may result in dermal and respiratory exposures sufficient to cause measurable toxicological responses in infants. While such exposures are not likely to produce recognizable symptoms of cholinesterase inhibition, the possibility of cases with subclinical effects cannot be ruled out. Confirmation of such exposures would require biological monitoring (e.g., detection of 3,5,6-trichloro-2-pyridinal in the urine for chlorpyrifos).

The potential for exposures greater than those reported here should also be noted. The training of many urban pesticide applicators is minimal and surveillance of application performance is nonexistent in most states. It is not unreasonable to assume that accidents and cases of inadvertent over-application will occur under these circumstances, albeit with low frequency. Furthermore, repeated applications for fleas and other household pests are not uncommon.

Current federal regulatory requirements for indoor pesticide applications are limited to label instructions. The label for broadcast applications of the compound under study here is typical (K. Lunchick, USEPA; personal communication); i.e., the premises should be vacated during application, and humans or pets should not contact treated surfaces until the spray has dried. These instructions are normally communicated by the applicator to occupants as a 1–2 hour reentry period. The finding of elevated air concentrations 3–7 hours post-application and the potential for dermal contact indicate that this reentry period is inappropriate for infants and small children.

Several strategies are available to reduce the risks associated with indoor pesticide applications. First, notification requirements could be established to inform occupants of potential health risks and means for minimizing exposure. Recommendations for broadcast applications of acutely toxic compounds such as chlorpyrifos might include thorough ventilation prior to reentry regardless of seasonal conditions, and well-defined reentry periods beyond 1–2 hours with special warnings regarding contact with treated surfaces by infants and small children. Second, more rigorous training and licensing procedures for applicators and application surveillance programs could be developed. Finally, product registrations could be modified or withdrawn for specific applications if an acceptable level of risk cannot be demonstrated.

ACKNOWLEDGMENTS

A preliminary report of this work was presented at the American Industrial Hygiene Conference, San Francisco, CA, May 15–20, 1988. The authors wish to thank Drs. Molly Coye, Michael Gallo, Philip Landrigan, Thomas Ledoux, and Mark Robson for their review of this manuscript. Special acknowledgment to David Hackathorn and Delmont Eberhart for their early insight into this field of investigation. The technical assistance of William Carey and Western Termite and Pest Control of New Jersey was greatly appreciated. This work was supported by State funds (NJAES Pub. No. D-07124-1-89).

REFERENCES

1. National Academy of Sciences: Urban Pest Management. Washington, DC: National Academy Press, 1980.
2. Gold RE, Holcslaw T, Tupy D, Ballard JB: Dermal and respiratory exposure to applicators and occupants of residences treated with dichlorvos (DDVP). J Econ Entomol 1984; 77:430–436.
3. Woody RC: The clinical spectrum of pediatric organophosphate intoxications. Neurotoxicology 1984; 5:75.
4. Zwiener RJ, Ginsburg CM: Organophosphate and carbamate poisoning in infants and children. Pediatrics 1988; 81:121–126.
5. Knaak JB, Schreider J, Berteau P: Hazard assessment of indoor use of chlorpyrifos, dichlorvos, propoxur and other organophosphates and N-methyl carbamates. Worker Health and Safety Branch Report No. HS-1423. Sacramento, CA: California Department of Food and Agriculture, 1987.
6. Berteau PE, Knaak JB, Mengle DC: Insecticide absorption from indoor surfaces: hazard assessment and regulatory requirements. *In:* Wang RGM,

Franklin CA, Honeycutt RC, Reinert JC (eds): Biological Monitoring for Pesticide Exposure, ACS Symposium Series 382. Washington, DC: American Chemical Society, 1989.

7. Lowengart RA, Peters JM, Cicioni C, Buckley J, Bernstein L, Preston-Martin S, Rappaport E: Childhood leukemia and parents' occupational and home exposures. JNCI 1987; 79:39–46.
8. Livingston JM, Jones CR: Living area contamination by chlordane used for termite treatment. Bull Environ Contam Toxicol 1981; 27:406.
9. Wright CG, Leidy RB: Chlordane and heptachlor in the ambient air of houses treated for termites. Bull Environ Contam Toxicol 1982; 20:617.
10. Wright CG, Leidy RB, Dupree HE, Sheets TJ: Subterranean termite control: chlordane residues in soil surrounding and air within houses. *In:* Honeycutt RC, Zweig G, Ragsdale NN (eds): Dermal Exposure Related to Pesticide Use, ACS Symposium Series 273. Washington, DC: American Chemical Society, 1985.
11. Moye HA, Malagodi MH: Levels of airborne chlordane and chlorpyrifos in two plenum houses: saranex S-15 as a vapor barrier. Bull Environ Contam Toxicol 1987; 39:533–540.
12. Anderson DJ, Hites RA: Chlorinated pesticides in indoor air. Environ Sci Technol 1988; 22:717–720.
13. Wright CG, Leidy RB, Dupress HE: Chlorpyrifos in the ambient air of houses treated for termites. Bull Environ Contam Toxicol 1988; 40:561–568.
14. Gillet JW, Harr JR, Linstrom FT, Mount DA, St. Clair AD, Weber LJ: Evaluation of human health hazards on use of dichlorvos (DDVP), especially in resin strips. Residue Rev 1972; 44:115–159.
15. Gillet JW, Harr JR, St. Clair AD, Weber LJ: Comments on the distinction between hazards and safety in evaluation of human health hazards on use of dichlorvos, especially in resin strips. Residue Rev 1972; 44:161–184.
16. Leary JS, Keane WT, Fontenot C, Feictmeir ES, Schultz D, Koos DA, Hirsch L, Lavor EM, Roan CC, Hine CH: Safety evaluation in the home of polyvinyl chloride resin strip containing dichlorvos (DDVP). Arch Environ Health 1974; 29:308–314.
17. Wright CG, Jackson MD: Propoxur, chlordane and diazinon on porcelain china saucers after kitchen cabinet spraying. J Econ Entomol 1971; 64:457.
18. Wright CG, Jackson MD: Insecticide residues in non-target areas of rooms after two methods of crack and crevice application. Bull Environ Contam Toxicol 1975; 13:123.
19. Wright CG, Jackson MD: Insecticide movement following application to crevices in rooms. Arch Environ Contam Toxicol 1976; 4:492.
20. Wright CG, Leidy RB: Chlorpyrifos residues in air after application to crevices in rooms. Bull Environ Contam Toxicol 1978; 19:340.
21. Wright CG, Leidy RB, Dupree HE: Insecticides in the ambient air of rooms following their application for control of pests. Bull Environ Contam Toxicol 1981; 20:548.
22. Wright CG, Leidy RB, Dupree HE: Chlorpyrifos and diazinon detection on surfaces in dormitory rooms. Bull Environ Contam Toxicol 1984; 32:259–264.
23. Hsu JP, Wheeler HG, Camann DE, Schattenberg HJ, Lewis RG, Bond AE: Analytical methods for detection of nonoccupational exposure to pesticides. J Chromatogr Sci 1988; 26:181–189.
24. Maddy KT, Lowe J, Saini N: Indoor air concentration of ethylene glycol monoethyl ether following application of propetamphos insecticide emulsifiable concentrate. Report No. HS-1264. Sacramento, CA: Worker Health and Safety Unit, CA Department of Food and Agriculture, 1985.
25. Naffziger DH, Sprenkel RJ, Mattler MP: Indoor environmental monitoring of Dursban L.O. following broadcast application. *In:* Down to Earth, No. 41, Midland, MI: Dow Chemical Company, 1985.
26. Maddy KT, Edmiston S, Fredrickson AS: Monitoring Residues of DDVP in room air and on an horizontal surface following use of a room fogger. Report No. HS-897. Sacramento, CA: Worker Health and Safety Unit, CA Department of Food and Agriculture, 1981.
27. Maddy KT, Edmiston S, Ochi E: Dissipation of DDVP and propoxur following the release of an indoor fogger—a preliminary study. Report No. HS-1259. Sacramento, CA: Worker Health and Safety Unit, CA Department of Food and Agriculture, 1984.
28. Kreiger R, Ross J, Thongsinthusak T, Fong H: Measuring potential dermal transfer of surface pesticide residue generated from indoor fogger use. Worker Health and Safety Branch Report. Sacramento, CA: California Department of Food and Agriculture, 1988.
29. Health and Welfare Canada: Guidelines for Indoor Occupant Exposure Assessment following Pesticide Applications in Indoor Environments. Ottawa, Ontario: Pesticides Division, Department of Health and Welfare, 1989.
30. Occupational Safety and Health Administration: Sampling for surface contamination. *In:* Industrial Hygiene Technical Manual, Chapter VIII, US Department of Labor. Washington, DC: US Government Printing Office, 1984.
31. National Academy of Sciences: An assessment of health risks of seven pesticides used for terminate control. National Research Council Committee on Toxicology. Washington, DC: National Academy Press, 1982.
32. National Center for Health Statistics: NCHS Growth Charts. Monthly Vital Statistics Report, 25(3), Suppl (HRA) 76-1120. Rockville, MD: Health Resources Administration, 1976.
33. US Environmental Protection Agency Office of Health and Environmental Assessment: Development of statistical distributions or ranges of standard factors used in exposure assessments. Springfield, VA: National Technical Information Service PB85-242667/AS, 1985.
34. Nolan RJ, Rick DL, Freshour NL, Saunders JH: Chlorpyrifos: pharmacokinetics in human volunteers. Toxicol Appl Pharmacol 1984; 73:8–15.
35. Fenske RA, Leffingwell JT, Spear RC: A video imaging technique for assessing dermal exposure—I. instrument design and testing. Am Ind Hyg Assoc J 1986; 47:764–770.
36. Fenske RA, Wong SM, Leffingwell JT, Spear RC: A video imaging technique for assessing dermal exposure—II. fluorescent tracer testing. Am Ind Hyg Assoc J 1986; 47:771–775.
37. Feldmann RJ, Maibach HI: Percutaneous penetration of some pesticides and herbicides in man. Toxicol Appl Pharmacol 1974; 28:126–132.
38. Noonan PK, Wester RC: Percutaneous absorption of nitroglycerin. J Pharm Sci 1980; 69:365–366.
39. Wester RC, Maibach HI: *In vivo* percutaneous absorption. *In:* Marzulli FN, Maibach HI (eds): Dermatotoxicology, Third Edition. Washington, DC: Hemisphere, 1987.
40. Shah PV, Fisher HL, Sumler MR, Monroe RJ, Chernoff N, Hall LL: Comparison of the penetration of 14 pesticides through the skin of young and adult rats. J Toxicol Environ Health 1987; 21:353–366.
41. FAO/WHO: 1972 evaluations of some pesticide residues in foods. WHO Pesticide Residue Series No. 2. Geneva: World Health Organization, 1973.
42. US Environmental Protection Agency: Integrated risk information system data base (IRIS). Washington, DC: USEPA, 1988.

'Late Breaker' Session on Injury Control Invites Abstracts

The Injury Control and Emergency Health Services special primary interest group of the American Public Health Association has announced it will again feature a "late breaker" session during the APHA upcoming 118th annual meeting in New York City. The session will be held on Tuesday, October 2, 8:30 am–10:00 am, and will feature work completed within the last few months—after the deadline for consideration in the regular symposia of the APHA annual meeting.

Abstracts of 250 words or less will be accepted by the Injury Control SPIG until August 15, 1990. Please send the abstract, title of the paper, authors' name, address and telephone number to: Richard Waxweiler, Division of Injury Control, Centers for Disease Control, Mail Stop F-36, Atlanta, GA 30333. Tel: 404/488-4695.

Breastfeeding Patterns in Puerto Rico

JOSE E. BECERRA, MD, MPH, AND JACK C. SMITH, MS

Abstract: In the 1950s, Puerto Rico began an active industrialization program. We used data from the 1982 Puerto Rico Fertility and Family Planning Assessment to describe the trend in the incidence of breastfeeding in Puerto Rico over time and to ascertain some of its determinants. From 1946 through 1982, 5,884 infants were born among this statistically representative sample of reproductive-aged women. The proportion of infants who had ever been breastfed was 59 percent for births before 1960 (mean duration = 7.8 months), dropped to 25 percent for infants born from 1970 to 1974 (mean duration = 4.9), and rose to 38 percent for births delivered from 1980 to 1982 (mean duration = 3.4). Prior breastfeeding experience was an important determinant of breastfeeding a newborn. Infants of mothers who had breastfed a previous baby were 7.3 times more likely to be breastfed (95% confidence interval = 6.6, 8.0) compared with infants of mothers who had not previously breastfed. The 38 percent of infants who were breastfed in Puerto Rico in the early 1980s is below the 74 percent to 97 percent reported in Latin America and below the 54 percent reported in the United States for the same period. This study provides baseline data for any future intervention strategies. (*Am J Public Health* 1990; 80:694–697.)

Introduction

A resurgence in the practice of breastfeeding has occurred throughout the world.[1] In the United States the downward trend in breastfeeding practice has been reversed since the late 1970s.[2] Breastfeeding trends vary in less developed countries, and three basic phases of progression have been described.[1]

The traditional phase is characterized by a high proportion of breastfed infants and a long duration of breastfeeding. In the transformation phase, both the proportion of breastfed infants and the duration of breastfeeding show a decrease. The third, or resurgent phase, is characterized by an increase in both the proportion of breastfed infants and the duration of breastfeeding. The patterns of these trends are not uniform in any country; they vary by geography and by the socioeconomic status of different groups.

The relation between the economic development initiated in Puerto Rico in the 1950s and breastfeeding practices among Puerto Ricans has not been studied before. The present study does so, using data from a 1982 survey.

Methods

In 1982, a multipurpose island-wide social, demographic, and maternal/child health study of women ages 15–49 years old living in Puerto Rico was undertaken as a collaborative project involving the University of Puerto Rico School of Public Health, the Puerto Rico Department of Health, and the Centers for Disease Control. The sampling methods have been described in detail elsewhere.[3] The completion rate of the household questionnaire was 91 percent.

A total of 3,175 women were interviewed and, of this number, 2,012 had had at least one live birth. For each live birth reported, mothers were asked: 1) "When your baby was an infant, did you breastfeed him/her?"; 2) "How many months old was s/he when you stopped breastfeeding him/her?"; and 3) "At what age was s/he first given any infant formula, cow's milk, solid foods other than breastmilk on a regular basis?" Of the 2,012 mothers queried, only one mother could not provide a response to any of these questions.

The total number of live births among the women in the sample was 5,884, all delivered between 1946 through 1982. For our analysis, we divided the study period into six birth delivery periods: 1946–59 (N = 709); 1960–64 (N = 924); 1965–69 (N = 1,135); 1970–74 (N = 1,239); 1975–79 (N = 1,207); and 1980–82 (N = 640). We were unable to determine the year of birth for 30 (0.5 percent) infants. Given the blurred distinction between rural and urban areas in Puerto Rico throughout the study period, the place where the delivery occurred (home, private hospital, and public hospital) was used instead of geographical (urban vs rural) correlates.

We used SESSUDAN,[4] a standard errors program, to compute standardized rates from sample survey data and to produce weighed estimates with 95% confidence intervals. We then calculated the proportion of infants who were ever breastfed and the proportion of infants who were exclusively breastfed for at least the first month of life for each birth delivery period stratified by place of delivery (home, private hospital, and private hospital) and by years of maternal education (<12, 12–15, >15) reported at the time of the interview (1982).

We also used the life table analysis program in BMDP[5] to calculate the mean duration of breastfeeding for each birth delivery period and for the three categories of maternal education for births occurring between 1975 and 1982. Infants of mothers still breastfeeding at the time of the interview and infants who had died while still breastfeeding were considered (statistically) censored observations.

We also used SAS[6] to calculate the breastfeeding percent ratio of multiparous mothers with and without previous breastfeeding experience, adjusted for years of maternal education and birth delivery period. In addition, we used the logistic regression program in BMDP[7] to simultaneously determine the strength of association (measured by the odds ratio [OR]) between the infant's breastfeeding status (ever breastfed or not) and maternal age in years (<20, ≥20), place where the mother was raised (continental United States, Puerto Rico), and years of maternal education (<12, 12–15, >15) among primiparas delivering from 1975 to 1982.

Results

In Puerto Rico, the proportion of infants who were ever breastfed was 37.3 percent (95% CI = 34.6, 39.9) for the entire period of 1946 to 1982 (Table 1). This proportion was

---

Address reprint requests to Jose E. Becerra, MD, MPH, Medical Epidemiologist, Pregnancy Epidemiology Branch, Division of Reproductive Health (CO6), Center for Chronic Disease Prevention and Health Promotion, Centers For Disease Control, Atlanta, GA 30333. Mr. Smith is Chief, Research and Statistics Branch, in that same Division at CDC. This paper, submitted to the Journal May 17, 1989, was revised and accepted for publication October 27, 1989.

TABLE 1—Trends for Infants Ever Breastfed in Puerto Rico (aggregated by parity), 1946–82

	Period of Birth						
	1946–59	1960–64	1965–69	1970–74	1975–79	1980–82	Total*
	Percent (sample size)						
Primiparous Mothers							
Ever breastfed	57.7 (312)	45.1 (313)	32.7 (393)	20.3 (405)	26.0 (391)	43.4 (193)	35.7 (2012)
Education (years)							
<12	62.8 (213)	48.8 (163)	37.3 (170)	17.9 (168)	20.7 (113)	32.0 (75)	39.1 (906)
12–15	50.5 (78)	39.4 (122)	27.5 (183)	21.4 (187)	28.0 (222)	44.8 (93)	31.8 (886)
>15	32.2 (21)	48.0 (28)	35.6 (40)	24.4 (50)	29.5 (56)	72.7 (25)	36.9 (220)
Type of facility							
Public hospital	62.4 (165)	48.0 (174)	36.7 (207)	18.4 (223)	21.7 (223)	42.6 (117)	36.0 (1112)
Private hospital	44.2 (94)	39.4 (127)	27.2 (179)	22.4 (179)	32.2 (167)	45.2 (75)	32.6 (821)
Home	68.8 (51)	— (9)	— (5)	— (2)	— (0)	— (0)	67.9 (69)
Multiparous mothers							
Ever breastfed	60.7 (397)	50.1 (611)	39.6 (742)	27.5 (834)	28.9 (814)	35.9 (446)	38.0 (3869)
Education (years)							
<12	62.0 (307)	55.5 (416)	44.2 (442)	30.2 (450)	28.8 (394)	30.8 (194)	41.7 (2224)
12–15	60.1 (74)	38.2 (156)	35.8 (241)	23.6 (314)	28.2 (345)	40.3 (197)	33.3 (1331)
>15	38.9 (16)	38.5 (39)	20.7 (59)	26.9 (70)	32.5 (75)	39.5 (55)	31.2 (514)
Type of facility							
Public hospital	58.2 (186)	51.2 (329)	43.7 (412)	30.6 (449)	30.0 (459)	33.8 (243)	38.9 (2087)
Private hospital	43.8 (89)	32.7 (186)	29.8 (282)	23.5 (364)	28.2 (338)	38.9 (199)	30.4 (1460)
Home	78.8 (116)	81.7 (86)	59.3 (44)	30.5 (17)	24.6 (11)	— (2)	68.9 (290)
Total*	59.4 (709)	48.4 (924)	37.3 (1135)	25.1 (1239)	28.1 (1207)	38.3 (640)	37.3 (5884)

(*)the difference between the sample size for total and the summation of cells corresponds to the missing values in each category.

59.4 percent (95% CI = 53.8, 65.0) for births occurring before 1960, dropped to 25.1 percent (95% CI = 21.2, 29.1) for infants born from 1970 to 1974, and rose to 38.3 percent (95% CI = 34.1, 42.4) for births delivered from 1980 to 1982 (Figure 1). The proportion of infants who were exclusively breastfed for at least the first month decreased from 27.4 percent (95% CI = 23.2, 31.3) before 1960 to 4.1 percent (95% CI = 0.0, 7.9) in 1982 (Figure 1).

The mean duration of breastfeeding for all infants throughout the study was 5.6 months (95% CI = 5.2, 5.9). The mean duration of breastfeeding declined consistently from 7.8 months (95% CI = 6.9, 8.6) for births occurring before 1960 to 3.4 months (95% CI = 2.8, 4.0) for births delivered from 1980 to 1982.

Breastfeeding status and place of delivery were related (Figure 2). Before 1975, infants delivered at home or in public hospitals were more likely to be breastfed than were infants delivered at private hospitals. However, this pattern is reversed after 1975. From 1980 to 1982, 40.6 percent (95% CI = 33.8, 47.5) of infants delivered in private hospitals were ever breastfed, compared with 36.8 percent (95% CI = 30.8, 42.8) of infants delivered in the public sector.

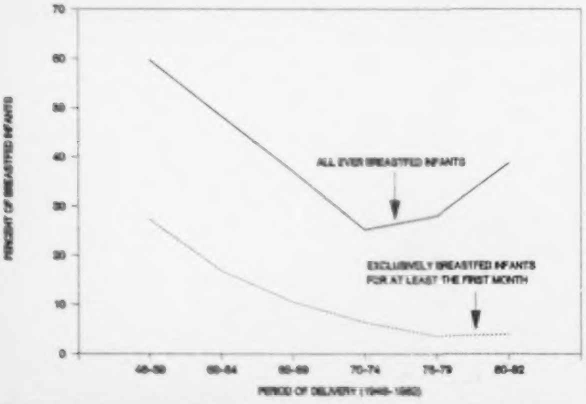
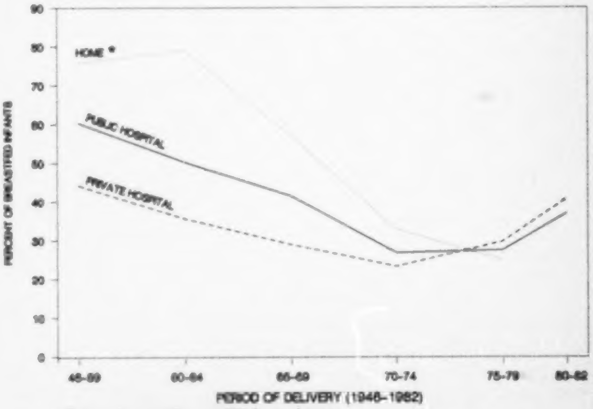


FIGURE 1—Breastfeeding Trends in Puerto Rico, 1946–82: Percent of Infants Breastfed by Birth Delivery Period



* Estimates for home births are unstable for recent years

FIGURE 2—Infants Ever Breastfed by Place of Delivery, Puerto Rico, 1946–82

A similarly reversed pattern appears in the practice of breastfeeding after 1975 in relation to maternal education (Figure 3). From 1980 to 1982, 50.0 percent (95% CI = 37.9, 61.6) of infants of mothers with more than 15 years of formal education were ever breastfed, compared with 41.7 percent (95% CI = 36.4, 47.1) for infants of mothers with 12–15 years of education, and 31.4 percent (95% CI = 25.1, 37.8) for infants of mothers with fewer than 12 years of education. However, multiparous mothers with fewer than 12 years of education did not show the same pattern as their primiparous counterparts in recent years (Table 1). From 1980–82, primiparous mothers showed the strongest correlation between

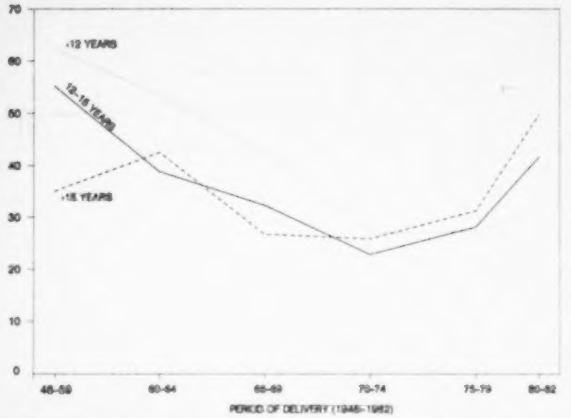


FIGURE 3—Infants Ever Breastfed and Maternal Education at Interview, Puerto Rico, 1946–82

education and breastfeeding practice and the greatest rise in the proportion of infants ever breastfed (Table 1).

From 1975 to 1982, the mean duration of breastfeeding for infants of mothers with more than 15 years of formal education and delivered after 1975 as 3.2 months (95% CI = 2.4, 4.1); the duration was 3.4 months (95% CI = 2.8, 3.9) for mothers with 12–15 years of education and 4.1 months (95% CI = 3.2, 4.9) for mothers with fewer than 12 years.

On the average, infants of multiparous mothers who had breastfed a previous baby were 7.3 times more likely to be breastfed (95% CI = 6.6, 8.0) than were infants of mothers who had not previously breastfed. This measure of association was adjusted for maternal education and birth delivery period.

The place where the mother had been raised, maternal education, and maternal age were examined among primiparas. From 1975 to 1982, maternal education was the strongest independent correlate of breastfeeding among first live-birth infants. Mothers with 12–15 years of education had OR of 1.8 (95% CI = 1.1, 2.8), and mothers with more than 15 years of formal education had OR of 2.8 (95% CI = 1.5, 5.1) compared with mothers with fewer than 12 years of formal education. These estimates were adjusted for maternal age and place where the mother was raised (this latter as a proxy for cultural identification).

Discussion

In the 1950s, Puerto Rico began an active industrialization program that reduced infant mortality from 114.1 infant deaths per 1,000 live births in 1940 to 44.4 in 1960 and 19.2 in 1980.[8] The life expectancy increased from 46.0 years in 1940 to 69.4 in 1960 and 74.9 in 1980.[8]

Our results document that the traditional phase of high rates of breastfeeding among women who delivered at home or in public hospitals and who were less educated declined in Puerto Rico with the advent of industrialization in the 1950s. By 1980, Puerto Rico was between the transformation and resurgent phases of breastfeeding practice. That is, the upward trend in the proportion of infants who were ever breastfed since 1975 was accompanied by a very low and decreasing proportion of infants who were exclusively breastfed during the first month of life. This trend shows that an increasing proportion of mothers who breastfed are probably using formula supplementation during the first month. However, very few medical indications justify introducing food other than breastmilk to a full-term baby during its first four months of life.[9]

Evidence shows that breastfeeding practice in Puerto Rico lags behind the United States and many Latin American countries. For 1980, 54 percent of infants in the United States had ever been breastfed[10] and, for the same period, 74 percent to 97 percent of all infants in selected Latin American countries had ever been breastfed.[11] In comparison, approximately 40 percent of infants born in Puerto Rico in 1980 had ever been breastfed. For Puerto Ricans living in the northeastern region of the United States, the proportion of infants who were ever breastfed is even lower: 26 percent for the 1980–82 period.*

The average number of months that an infant is breastfed in Puerto Rico is consistently decreasing. As previous research has shown in other populations,[12,13] the lack of adequate familial, social, and professional support, and the insensitive employment environment facing modern, highly educated women discourage mothers from breastfeeding. The downward trend in number of months of breastfeeding parallels the decreasing proportion of infants exclusively breastfed for at least the first month of life.

The association between maternal education and breastfeeding is reversed during the upward trend of breastfeeding after 1975. This reversal is consistent with previously published studies.[2] After 1975, mothers with higher education were more likely to initiate breastfeeding than were mothers with less education, whereas the opposite was true before 1960. This pattern is similar to the association between place of delivery and breastfeeding. It is important to realize that institution of delivery is associated with maternal education and socioeconomic status in Puerto Rico.[3]

Although recall bias could be invoked to explain some of these findings, we find no plausible and consistent hypothesis to support such contention. In addition, the general pattern of our results follows the expected interaction between economic development and breastfeeding practices.

Since 1975, maternal education, probably measuring (in addition to socioeconomic status) health behavior awareness and increased capacity to change health belief systems, is strongly correlated with initiation of breastfeeding among primiparas. This trend is encouraging and should guide policy-makers to carry out interventions that will improve breastfeeding practices on the island. However, the decreasing duration of breastfeeding in recent years as maternal education increases provides grounds for concern. This finding differs with previously published studies[14] on the relationship between maternal education and duration of breastfeeding in recent years.

Previous breastfeeding experience was a very strong predictor of breastfeeding a subsequent infant in our study. This fact underscores the importance of role models for young mothers and the self-sustaining nature of a change in breastfeeding behavior. This finding held true even after we adjusted for maternal education and birth delivery period.

In addition to the well known psychological and biological benefits of breastfeeding for the infant,[15] intervention strategies should also emphasize the maternal benefits of breastfeeding: breastfeeding helps postpartum weight loss,[16] breastfeeding prolongs postpartum amenorrhea,[17] and breastfeeding may exert a protective effect against premenopausal breast cancer.[18,19]

---

*Carter O, Ezzati T: Breastfeeding practices among Mexican Americans, Cuban Americans, and Puerto Ricans: HHANES, 1982–84 (Abstract). American Public Health Association 115th Annual Meeting, 1987, New Orleans, LA.

In summary, breastfeeding practice in Puerto Rico in 1982 fell between the transitional and resurgent phases of progression. Although we have documented an upward trend in the proportion of infants who were ever breastfed since 1975, more sensitive indicators of breastfeeding practice, such as the proportion of infants who were exclusively breastfed in the first month and duration of breastfeeding, show disturbing decreasing trends in Puerto Rico.

ACKNOWLEDGMENT

The authors wish to acknowledge the original contributions and thoughtful review of the manuscript made by Dr. Rosa B. Licha-de-Becerra. Part of this work was presented at the American Public Health Association, 115th Annual Meeting, October 19, 1987, New Orleans, Louisiana.

REFERENCES

1. Division of Family Health, World Health Organization: The prevalence and duration of breastfeeding: Critical review of available information. World Health Stat Q 1982; 35(2):92–112.
2. Hendershot GE: Trends in breastfeeding. Pediatrics 1984; (Suppl) 74 (Part 2):591–602.
3. Becerra JE, Smith JC: Maternal smoking and low birthweight in the reproductive history of women in Puerto Rico, 1982. Am J Public Health 1988; 78:268–272.
4. Shah BV: SESSUDAN: Standard errors program for computing of standardized rates from sample survey data. Research Triangle Park, NC: Research Triangle Institute, 1981.
5. Benedetti J, Yuen K, Young L: Life tables and survival functions. *In*: Dixon WJ (ed): BMDP Statistical Software. Los Angeles: University of California Press, 1983.
6. SAS Institute Inc: SAS User's Guide: Statistics, Version 5 Edition. Cary, NC: SAS Institute Inc, 1985; 403–432.
7. Engelman L: Stepwise logistic regression. *In*: Dixon WJ (ed): BMDP Statistical Software. Los Angeles: University of California Press, 1983.
8. Vasquez-Calzada JL: El descenso de la mortalidad en Puerto Rico: un hecho historico notable. P R Health Sci J 1984; 3:173–181.
9. American Academy of Pediatrics: Breastfeeding: a commentary in celebration of the year of the child, 1979. Pediatrics 1978; 62:591–601.
10. Martinez GA, Nalezienski JP: 1980 update: The recent trend in breastfeeding. Pediatrics 1981; 67:260–263.
11. Ferry B, Smith DP: Breastfeeding differentials. World Fertility Survey Comparative Studies (Cross National Summaries) 1983; 23:10.
12. Scrimshaw SC, Engle PL, Arnold L, Haynes K: Factors affecting breastfeeding among women of Mexican origin or descent in Los Angeles. Am J Public Health 1987; 77:467–470.
13. Bryant CA: The impact of kin, friend and neighbor network on infant feeding practices: Cuban, Puerto Rican and Anglo families in Florida. Soc Sci Med 1982; 16:1757–1765.
14. Simopoulos AP, Grave GD: Factors associated with the choice and durations of infant-feeding practice. Pediatrics 1984; (Suppl) 74 (Part 2):603–614.
15. Lawrence RA: Breastfeeding: A Guide for the Medical Profession. St. Louis: CV Mosby Company, 1985.
16. Brewer MM, Bates MR, Vannoy LP: Postpartum changes in maternal weight and body fat depots in lactating vs nonlactating women. Am J Clin Nutr 1989; 49:259–65.
17. Short RV: Breastfeeding. Sci Am 1984; 250:35–41.
18. Byers T, Graham S, Rzepka T, Marshal J: Lactation and breast cancer: evidence for a negative association in premenopausal women. Am J Epidemiol 1985; 121:664–674.
19. McTiernan A, Thomas DB: Evidence for a protective effect of lactation on risk of breast cancer in young women. Am J Epidemiol 1986; 124:353–358.

Nominations Invited for 1990 McAlpin Award in Mental Health Research

The National Mental Health Association (NMHA) is seeking nominations for its 1990 McAlpin Mental Health Research Achievement Award to be presented to a researcher in the field of mental illness. The award honors the memory of William R. McAlpin, Jr., who was devoted to the cause of mental health and the advancement of mental illness research.

Nominations will be considered by a panel of judges for researchers whose work offers significant promise in increasing scientific knowledge in the understanding of one or more of the mental illnesses and meets the highest research standards. Previous distinguished recipients are Donald F. Klein, MD, Columbia University; Floyd Elliott Bloom, MD, Salk Institute Center for Behavioral Neurobiology; Paul Greengard, PhD, Rockefeller University; and William T. Carpenter, Jr., MD Maryland Psychiatric Research Center.

The McAlpin Award will be presented during the Seventh Annual National Scientific Symposium on Mental Health and Mental Illnesses, to be held in Houston, Texas, September 14–15. The recipient will be expected to attend the symposium to accept the award.

Nominations from sponsors (individuals and organizations) must be received no later than July 31, 1990. For complete details on nomination procedures contact the NMHA Executive Office, 1021 Prince Street, Alexandria, VA 22314-2971, (703) 684-7722 for a brochure on the 1990 McAlpin Award.

Dietary Intake of Americans Reporting Adherence to a Low Cholesterol Diet (NHANES II)

GORDON SCHECTMAN, MD, W. PAUL MCKINNEY, MD, JOAN PLEUSS, RD, AND RAYMOND G. HOFFMAN, PHD

Abstract: We studied the dietary intake of persons age 18 and over participating in the 1976–80 National Health and Nutrition Examination Survey (NHANES II). We compared the diets of those on a "low cholesterol" diet (dieters, n = 296) with the diets of individuals not following a special diet (non-dieters, n = 10,052). Dietary intakes were assessed by 24-hour recall and food frequency reports and were adjusted for differences in age, sex, race, education, smoking, and socioeconomic status by multivariate statistical techniques. Dieters consumed 16 percent (99% confidence interval- −24, −9) fewer calories than non-dieters, and had decreased intake of saturated fat by 25 percent (−34, −15) and cholesterol by 21 percent (−32, −10). However, their intake of vitamin A, thiamine, riboflavin, niacin, calcium, and iron were not significantly different from non-dieters and their ascorbic acid intake was 18 percent (1, 36) higher. The nutrient density (nutrient/1000 kcal) among dieters was increased over non-dieters by 35 percent (−3, 72) for vitamin A, 35 percent (1, 70) for thiamine, 30 percent (−4, 65) for riboflavin, 27 percent (9, 44) for niacin, 60 percent (40, 80) for vitamin C, 14 percent for calcium (−5, 32), and 15 percent for iron (6, 25). Compared with non-dieters, dieters consumed 27 percent (9, 45) more poultry and 39 percent (12, 66) more fish, but consumed 30 percent (−44, −16) less eggs and 15 percent (−24, −6) less meat. (*Am J Public Health* 1990; 80:698–703.)

Introduction

Recent evidence has linked reduction of blood cholesterol with reduced incidence of coronary heart disease.[1,2] Dietary intervention effectively lowers blood cholesterol levels[3,4] and therefore the cholesterol lowering diet is currently emphasized as an important public health measure. Recent guidelines established by the National Cholesterol Education Program have defined blood cholesterol values above 200 mg/dl as undesirable due to their association with increased coronary heart disease risk[5]; more than half of all Americans fall into this category.[6] Community programs to educate the public concerning hypercholesterolemia and the importance of a proper diet have recently been established,[7-9] and many individuals now report dietary changes in response to these initiatives.[8] Because efforts to enhance knowledge concerning hypercholesterolemia and diet among the public are still in progress, one may anticipate that more Americans will attempt dietary modification in the near future.

Despite this willingness to change dietary habits, the ability of Americans who adopt a "low cholesterol diet" to make significant dietary modifications is unknown. Compared with the typical American diet, the cholesterol lowering diet recommended by the National Cholesterol Education Program is lower in total fat, saturated fat, and cholesterol. In addition, caloric intake is limited, particularly in overweight individuals.[5] To achieve these goals, foods must be substituted into the diet to replace those which are rich in saturated fat and cholesterol. To modify food intake correctly, the dieter must be aware of the basic principles of the cholesterol lowering diet as well as the nutritional adequacy of potential food substitutions. Otherwise, the modification of food choices may have minimal effect on saturated fat and/or cholesterol intake, or may be made at the expense of the intake of other important nutrients. Subjects recruited for research studies designed to evaluate the efficacy of a short period of dietary intervention have generally shown significant decreases in serum cholesterol levels when the diet was instituted, suggesting that appropriate dietary changes were implemented during the study period.[1-4,10,11] However, individuals participating in these studies were highly selected and were provided with intensive nutrition counseling; therefore, they may not be representative of most Americans who wish to adopt a cholesterol lowering diet.

The purpose of this study was to determine if Americans who adopt a cholesterol lowering diet make dietary modifications that: 1) are in accordance with accepted dietary guidelines, and 2) allow for an adequate supply of other nutrients.

Methods

Data were analyzed from the second National Health and Nutrition Examination Survey (NHANES II), a comprehensive population survey which included a detailed dietary questionnaire from which nutrient intakes were calculated in a sample of over 10,000 adults. The NHANES II sample was selected so that population subgroups of special interest for nutritional assessment were oversampled. Among adults, the oversampled groups included persons over 60 years of age and persons below the poverty level. Sample weights were then computed for each participant so that the total sample closely estimated the noninstitutionalized civilian population. This sample weighting reflects the individual selection probability, adjustments for non-response, and post-stratification adjustments. A complete description of the survey methods has been reported.[12]

Data from NHANES II were acquired from sample persons over several visits. At the initial visit, demographic data (i.e., age, education and financial status, household information, etc.) and some of the medical history were obtained. At the second visit, the remainder of the medical history, dietary recall and food frequency information, and blood for laboratory testing were acquired. Adults ages 18 to 74 who attended this second visit were included in this study. Because not all persons who were willing to undergo the initial visit agreed to participate at the second visit, the potential for bias was introduced if these persons differed significantly from other sample persons. The sample weighting in NHANES II was therefore adjusted to correct for this

---

From the Division of General Internal Medicine, the Clinical Research Center, and the Department of Epidemiology and Biostatistics, Medical College of Wisconsin. Address reprint requests to Gordon Schectman, MD, Division of General Internal Medicine, Medical College of Wisconsin, 8700 West Wisconsin Avenue, Milwaukee, WI 53226. This paper, submitted to the Journal April 21, 1989, was revised and accepted for publication October 18, 1989.

problem.[13] This sample weight (the "examined final weight") has been employed for all analyses in this paper.

Nutrient intakes for vitamin A, thiamin, riboflavin, niacin, vitamin A, iron, and calcium were estimated from 24-hour recall interviews. These nutrient values were obtained from the US Department of Agriculture reference handbook[14] or directly from the food manufacturer, and were recorded directly on the NHANES II data tapes following completion of the survey. Vitamin and mineral supplements were not included in the calculation of nutrient intake. If two major meals could not be recalled, or if the respondent refused to cooperate or was uncooperative due to attitude, then the recall data were judged to be unsatisfactory by the interviewer. These unsatisfactory interviews were excluded from the analysis; imputed dietary intakes were not substituted for these unsatisfactory recalls. Three-month food frequency reports provided estimates of consumption of specific food groups (such as skim and whole milk, eggs, meat, fish, poultry, etc.). Responses from food frequency questions were converted into the number of weekly servings; respondents reporting occasional intake of less than one serving weekly were arbitrarily assigned a weekly intake of 1/2 serving. Dietary recall data are presented in Tables 2 through 5; data from the food frequency reports are presented in Table 6.

All participants attending the second visit were asked the question "Are you following a special diet?" If they responded affirmatively, they were then asked a series of questions to define the diet, such as "Is this diet to lose weight?"; "Is this a low salt diet?"; or "Is this a vegetarian diet?" Additional questions asked included whether the diet was prescribed by a health professional and the length of time that the diet had been followed. The complete list of questions asked to describe the diet are provided in the Appendix. Participants who were told by a physician that they had a previous heart attack or "hardening of the arteries" were considered to have a history of coronary artery disease. The poverty index, provided in NHANES II, was used to assess socioeconomic status. This is a measure of financial income adjusted to geographic poverty level for family unit; values of less than 1.0 are below the poverty level.

Because the association of dietary cholesterol with blood cholesterol and coronary heart disease was widely recognized during the period when NHANES II was conducted,[15-23] this study evaluated the dietary patterns of individuals (n = 296) who responded affirmatively to the question "Are you following a low cholesterol diet?" We assumed that individuals following this diet were attempting dietary modification to reduce their blood cholesterol levels and hence their risk of coronary heart disease. Multiple responses to define the diet were permitted in the survey. For example, among subjects stating adherence to a "low cholesterol diet," 55 percent also reported following a "low calorie diet," 68 percent followed a low fat diet, 12 percent followed a "diabetic diet," and 54 percent followed a "low salt diet." Respondents who reported following other diets (i.e., high blood pressure, diabetes) in addition to the "low cholesterol" diet were included in this study.

TABLE 1—Characteristics of Study Participants

	Dieters[a]	Non-dieters[b]
N	296	10052
Age (SD)	50.3(14)	39.9(16)
Sex (% male)	49.7	44.7
Race (% White)	91.2	89.8
BMI[c] (SD)	26.6 (4.8)	24.9 (4.8)
History of CAD (%)	25	3
Smoking (%)	25	38
Poverty Index[d]	2.9 (1.5)	2.7 (1.5)
Grade Level[e]	12.2 (2.8)	12.0 (3.1)

[a] Dieters-Study participants who reported adherence to a low cholesterol diet.
[b] Non-dieters-Study participants who did not report adherence to a specific diet.
[c] Kg/m2.
[d] Poverty Index-financial income adjusted to geographic poverty level for family unit; values of less than 1.0 are below the poverty level.
[e] Grade Level-Highest grade completed.

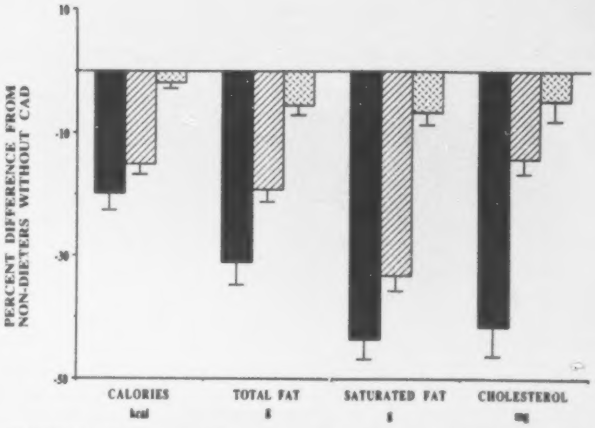


FIGURE 1—The Percent Difference in Caloric, Total Fat, Saturated Fat, and Cholesterol Intake of Dieters with CAD (black bars; N = 72), Dieters without CAD (hatched bars; N = 217), and Non-dieters with CAD (speckled bars; N = 323) compared to Non-dieters without CAD (N = 9681)

A total of 12,501 adults attended the second visit. However, respondents who were following special diets other than a low cholesterol diet (i.e., diabetic, hypertensive, low sodium, low calorie, n = 1,608), who did not have sufficient financial information recorded to compute a poverty index (n = 390), who did not record their level of education (n = 100), or who were judged by the interviewer to have unsatisfactory 24-hour recalls (n = 55) were excluded from the analysis. After these exclusions, 10,348 respondents remained eligible for the study. Those who attended the second visit but were subsequently excluded (n = 2,153) were more likely than included subjects to be male (49.2 vs 38.3 percent), but were otherwise similar to included subjects in percent who were White (89.0 vs 86.4 percent), not following a low cholesterol diet (97.4 vs 96.7 percent), and in their mean age (50.9 vs 45.2 years).

Individuals who were not adhering to any special diet (n = 10,052) were then compared with participants who indicated that they were following the low cholesterol diet (n = 296). In this paper, respondents who reported adherence to the low cholesterol diet are referred to as "dieters"; those subjects not following a diet are referred to as "non-dieters."

Statistical Analysis

Variance estimates were corrected for the complex sample design using the SUPERCARP program.[24] Multiple regression[25] was used to determine the effect of diet status (i.e., dieter compared with non-dieter) on the dietary intake of each nutrient or food group, after adjusting for age, gender, educational level, poverty index, race, and number of cigarettes smoked daily. The body mass index (BMI) was not used

TABLE 2—Intake of Protein, Carbohydrate and Fat among Americans Reporting Adherence to a Low Cholesterol Diet (Dieters) Compared with Americans not Following a Specific Diet (Non-dieters)

Nutrients	Non-Dieters Intake	Dieters Intake	Percent Difference Dieters vs Non-Dieters (99% CI)
Calories (kcal)	2072	1735	−16.3 (−23.7, −8.9)
Protein (g)	80.2	73.7	−8.0 (−15.8, −0.3)
Carbohydrate (g)	225.6	196.5	−12.9 (−21.1, −4.7)
Fat (g)	85.4	66.4	−22.1 (−30.8, −13.3)
Saturated (g)	30.7	23.2	−24.5 (−33.7, −15.4)
Monounsaturated (g)	31.6	24.2	−23.3 (−33.7, −12.8)
Polyunsaturated (g)	11.9	9.7	−18.6 (−30.1, −7.2)
Cholesterol (mg)	363	287	−20.8 (−31.8, −9.7)

TABLE 3—Percent of Total Caloric Intake for Protein, Carbohydrate and Fat among Americans Reporting Adherence to a Low Cholesterol Diet (Dieters) Compared with Americans not Following a Specific Diet (Non-dieters)

	Percent of Total Caloric Intake		
Nutrients	Non-Dieters Intake	Dieters Intake	Percent Difference Dieters vs Non-Dieters (99% CI)
Protein	15.6	18.3	16.8 (6.8, 26.8)
Carbohydrate	44.6	46.4	4.1 (0.3, 7.8)
Total Fat	36.5	32.8	−9.9 (−13.5, −6.3)
Saturated Fat	13.0	11.1	−14.4 (−19.7, −9.1)
Monounsaturated Fat	14.9	12.9	−13.6 (−19.0, −8.2)
Polyunsaturated Fat	5.8	5.5	−3.8 (−15.3, 7.8)

TABLE 4—Mean Intake of Vitamins and Minerals among Americans Reporting Adherence to a Low Cholesterol Diet (Dieters) Compared with Americans not Following a Specific Diet (Non-dieters)

Nutrients	Non-Dieters Intake	Dieters Intake	Percent Difference Dieters vs Non-Dieters (99% CI)
Vitamin A (IU)	5249	5284	+0.7 (−25.6, 27.0)
Thiamin (mg)	1.33	1.38	+4.3 (−15.0, 23.7)
Riboflavin (mg)	1.84	1.82	−1.2 (−17.9, 15.4)
Niacin (mg)	21.1	20.4	−3.1 (−11.4, 5.2)
Ascorbic acid (mg)	100.0	118.3	+18.3 (0.4, 36.2)
Calcium (mg)	778	688	−11.5 (−23.4, 0.4)
Iron (mg)	13.7	12.5	−9.0 (−18.4, 0.4)

TABLE 5—Mean Vitamin and Mineral Nutrient Density (nutrient/1000 kcal) of Americans Reporting Adherence to a Low Cholesterol Diet (Dieters) Compared with Americans not Following a Specific Diet (Non-dieters)

Nutrient Density (nutrient/1000 kcal)	Non-Dieters Intake	Dieters Intake	Percent Difference Dieters vs Non-Dieters (99% CI)
Vitamin A (IU/1000 kcal)	2818	3790	+34.5 (−3.4, 72.4)
Thiamin (mg/1000 kcal)	0.66	0.89	+35.0 (0.7, 69.7)
Riboflavin (mg/1000 kcal)	0.91	1.18	+30.3 (−4.2, 64.9)
Niacin (mg/1000 kcal)	10.59	13.42	+26.7 (9.1, 44.4)
Ascorbic acid (mg/1000 kcal)	52.7	85.9	+60.1 (39.8, 80.4)
Calcium (mg/1000 kcal)	381	433	+13.8 (−4.9, 32.4)
Iron (mg/1000 kcal)	6.89	7.95	+15.4 (6.2, 24.6)

as a covariate in the analyses presented in this paper because of the potential effect that diet status may have on body weight. However, separate analyses were also performed in which the BMI was included as an additional covariate. All covariates were forced into the model. Dependent variables not approximating a normal distribution were transformed logarithmically; these included saturated fat, milk, poultry, organ meats, vitamins A and C, and the nutrient density of vitamins A, B_1, B_2, and iron. However, the results of the analysis were not affected by this transformation and, for purposes of clarity and consistency, the data are presented untransformed. The Cook's distance was determined for residuals and found to be less than 0.2, indicating that outliers did not significantly bias the results of the analyses.[26] In Tables 2, 3, 4, and 6, the adjusted mean values for both non-dieters and dieters are given, as well as the percent difference between the dieters and non-dieters. Because of the large number of analyses performed, the 99% CI, rather than the 95% CI, was employed in this paper and is provided in the tables; negative values indicate that the mean intake for dieters is lower than the intake for non-dieters.

Results

Characteristics of dieters are compared with non-dieters in Table 1. Dieters were older, more likely to be female, and have slightly lower BMI than non-dieters, but were similar in poverty index, education level, and race. Dieters were also more likely to have coronary artery disease and less likely to be smokers. The mean duration that the diet had been followed was 147 weeks. A diet prescription was received from a health professional (dietitian, nurse or doctor) by 69 percent of dieters.

Total caloric intake was 16 percent lower (99% CI = −24, −9) in dieters compared with non-dieters, representing a decrease of 340 kcal (Table 2). Although part of this decrease was due to reduced protein and carbohydrate intake among dieters, most of the reduction in total calories was due to a 22 percent (99% CI = −31, −13) decrease in total fat intake. This reduction in total fat intake resulted from lower saturated, monounsaturated, and polyunsaturated fat consumption. Mean cholesterol intake was also reduced in dieters compared with non-dieters (21 percent decrease; 99% CI = −32, −10).

The composition of the diet among dieters also differed compared to non-dieters (Table 3). Dieters had a higher percent of total calories obtained from protein and carbohydrate and a lower proportion of total calories derived from total fat, saturated fat, and monounsaturated fat. The percent of total calories derived from polyunsaturated fat was similar between dieters and non-dieters. Dieters were more likely to meet goal intake levels for the cholesterol lowering diet by restricting total fat to 30 percent of the diet, saturated fat to 10 percent of the diet, and cholesterol intake to 300 mg daily. For dieters, 35.2 percent met this goal for total fat compared with 22.3 percent for non-dieters, while 41.7 percent of dieters and only 23.2 percent of non-dieters met this criteria for saturated fat. For dietary cholesterol, 66.5 percent of dieters, compared with 54.3 percent of non-dieters, achieved the goal level for daily intake.

Because the presence of coronary artery disease (CAD) may also motivate individuals to consume more healthful foods without consciously following a cholesterol lowering diet, the influence of previous CAD on dietary intake was evaluated. Compared to non-dieters without CAD, dieters both with and without a previous history of CAD had decreased caloric, total fat, saturated fat, and cholesterol intake (Figure 1). On the other hand, non-dieters with a history of CAD had caloric, total fat, saturated fat, and cholesterol intake similar to non-dieters without a CAD history.

Despite the decreased caloric intake, dieter vitamin and mineral intake was not significantly reduced compared with non-dieters (Table 4). This finding occurred because dieters selected foods with increased nutrient density (nutrient/1000 kcal). Marked increases in nutrient density were observed in dieters compared with non-dieters for vitamin A, thiamin, riboflavin, ascorbic acid, and niacin (Table 5). Smaller increases in nutrient density for iron and calcium were observed, and the total intake of these minerals in dieters was slightly decreased.

Dieters selected food that conforms to dietary guidelines for a cholesterol lowering diet (Table 6). They consumed 45 percent less whole milk products and 60 percent more skim milk products than non-dieters. Dieters ate 30 percent fewer eggs than non-dieters, and consumed 15 percent less meat. Consumption of poultry and fish were significantly increased compared with non-dieters.

Subsequent analyses were performed using the BMI as an additional covariate. All differences between dieters and non-dieters persisted; inclusion of this variable as a covariate had very little effect on the results for any nutrient, suggesting that the observed differences between groups was not attributable to body mass.

Discussion

Americans reporting adherence to a cholesterol lowering diet had dietary patterns which differed markedly from study participants who were not following any diet. Dieters reduced their intake of calories, total fat, and saturated fat. They consumed less red meat and whole milk and consumed more poultry, fish, and skim milk. Despite these significant differences in diet between dieters and non-dieters, dieters maintained their intake of essential vitamins and minerals by selecting food with increased nutrient density. These dietary trends are in accordance with dietary guidelines designed to reduced blood cholesterol levels. The findings suggest that dieters had sufficient nutritional understanding to make

TABLE 6—Mean Servings from Specific Food Groups Consumed Weekly by Americans Reporting Adherence to a Low Cholesterol Diet (Dieters) Compared with Americans not Following a Specific Diet (Non-dieters)

Food Groups	Non-Dieters Servings/week	Dieters Servings/week	Percent Difference Dieters vs Non-Dieters (99% CI)
Whole milk products	4.55	2.52	−44.7 (−58.6, −30.8)
Skim milk products	2.92	4.66	+59.5 (22.9, 96.1)
Meat	6.53	5.58	−14.6 (−23.3, −5.9)
Organ meats (i.e. liver)	0.38	0.31	−19.2 (−34.6, −3.8)
Poultry	1.34	1.71	+27.0 (8.8, 45.2)
Fish	1.02	1.42	+39.1 (12.4, 65.7)
Eggs	2.68	1.88	−29.7 (−43.5, −15.9)

specific food choices to reduce dietary cholesterol, total fat, and saturated fat without compromising the nutrient intake of their overall diet.

Since NHANES II was conducted from 1976–80, this study reflects the prevailing dietary recommendations during that period. Many advances in the current understanding of the cholesterol lowering effect of specific nutrients have occurred within the past decade.[27] However, the dietary guidelines to lower blood cholesterol levels were formulated in the 1970s and included the recommendations for the reduction of total fat, saturated fat, and dietary cholesterol,[15,16,21–23] similar to those recommended currently.[5] Although the reported dietary intake of dieters was consistent with these recommendations, the extent that knowledge of these guidelines actually influenced and/or motivated dieters to improve their diets cannot be determined from a cross-sectional study of this nature.

It is of interest that 31 percent of the dieters did not receive a diet prescription from a health professional (dietitian, nurse or physician) and decided on their own to follow a cholesterol lowering diet. In fact, they adhered to the cholesterol lowering diet as well as individuals who had received a diet prescription (data not shown). This is important because individuals with desirable (<200 mg/dl) or borderline high blood cholesterol levels (200–239 mg/dl) may be less likely to be referred by a health professional for dietary counseling compared with persons with higher values.[28] Since even individuals with relatively desirable cholesterol levels may benefit from a cholesterol lowering diet, it is reassuring that many Americans can successfully follow this diet even without formal consultation with a dietitian.

This study is limited by the methodological problems associated with estimating dietary intake in cross-sectional studies of free-living populations. Dietary recall of the preceding 24-hour period may not provide an accurate or representative assessment of current nutritional intake for an individual. However, this limitation is lessened when assessing the average intake of a group, since the group mean intake is less sensitive to day-to-day variability.[29] Further error in the data can be introduced during the reporting and recording of food intake, as well as in the analysis of the nutrient content of food.[30] During NHANES II, efforts were made to reduce these sources of error by employing trained interviewers and using a uniform approach to nutrient analysis during the entire survey. Because data from both dieters and non-dieters were acquired using the same methodology, error introduced from these sources would not be likely to bias the results of this study. An additional concern is that questions concerning adherence to a special diet may have biased dietary recall data. For example, individuals who reported adherence to the low cholesterol diet may have underreported ingestion of foods high in cholesterol and saturated fat. However, the questions concerning adherence to specific diets were among hundreds asked of each respondent and were obtained by interviewers who were unaware of the goals of the current study. Therefore, it is unlikely that questions describing the diet biased the reporting of nutrient intake data.

The results of a cross-sectional study alone cannot define the presence of a causal relationship. For example, individuals who naturally prefer diets particularly low in cholesterol and fat may report that they are following a low cholesterol diet. These individuals may not have modified their food intake due to the adoption of a diet. However, this hypothesis is unlikely because dieters, compared with non-dieters, had a much higher prevalence of coronary heart disease, suggesting that they modified their diet in response to this illness. Further, 69 percent of dieters had received the diet prescription from a health professional. Therefore, a more likely explanation for our results is that dietary intake was modified as a consequence of the adoption of the diet.

Diet modification can effectively reduce blood cholesterol levels. Studies specifically designed to evaluate the efficacy of cholesterol lowering diets report cholesterol reductions in the range of 10–15 percent.[11,31–33] Further, the diet is well tolerated.[34] However, perfunctory and routine dietary instruction in patients without proper motivation may have no impact on dietary compliance, weight reduction, or blood cholesterol levels.[35,36] In our study, only subjects who reported that they were following a cholesterol lowering diet were included as dieters. Therefore, individuals prescribed a cholesterol lowering diet but who were unwilling or unable to comply would probably not report adherence to the low cholesterol diet at the time of the survey, and would be excluded from the analysis. Thus, this report is likely to include only respondents who were sufficiently motivated to adopt this diet, and who felt that they were, at least in part, successful. Only 3 percent of the total sample, including only 15 percent of those with coronary heart disease, reporting following the low cholesterol diet. The willingness of the American public to accept this diet will be one important factor determining general compliance and efficacy.

Results from this study indicate that adherents to the cholesterol lowering diet report consumption of foods and nutrients consistent with recommended cholesterol lowering diet guidelines. In addition, the intake of essential vitamins and minerals is not compromised by adherence to this diet. Since the NHANES II survey utilizes a population-based sample, subjects included in the analysis represent a cross-section of the American public. Therefore, this analysis suggests that favorable dietary modifications can be achieved by Americans who choose to follow this diet. Efforts to increase the American public's awareness of the importance of dietary intervention to reduce cholesterol levels and thereby decrease coronary heart disease risk should be emphasized. Once Americans are prepared to adopt the cholesterol lowering diet, these results suggest that healthful dietary modification in accordance with accepted guidelines to reduce blood cholesterol levels is an achievable goal.

APPENDIX

NHANES II Questions Asked to Describe Diet

	Response
Are you on a special diet?	Yes/No
If yes, is this diet to lose weight?	"
Gain weight?	"
For diabetes?	"
For kidney failure?	"
For ulcers?	"
For diverticulitis?	"
For allergies?	"
For heart trouble?	"
For high blood pressure?	"
For pregnancy? (females only)	"
For any other reason?	"
What kind of diet is it?	
	Yes/No
Low fat?	"
Low protein?	"
High protein?	"

APPENDIX, cont.

	Response Yes/No
Low salt?	"
Low carbohydrate?	"
Low sugar?	"
Low caloric?	"
Low cholesterol?	"
High caloric?	"
Vegetarian with animal by-products? (egg, dairy, etc.)	"
Vegetarian without animal by-products?	"
A bland diet?	"
Some other type?	"
Was this diet prescribed by a doctor, dietitian or nurse?	"
Length of diet (in weeks)	

ACKNOWLEDGMENTS

The authors wish to express their appreciation to Kathy Sobcinski for assisting with the data analysis. This paper was presented in part at the American Federation of Clinical Research, Midwest Section, November 1988.

REFERENCES

1. Lipid Research Clinics Program: The Lipid Research Clinics Coronary Primary Prevention Trial results. I. Reduction in incidence of coronary heart disease. JAMA 1984; 251:351–364.
2. Blankenhorn DH, Nessim SA, Johnson RL, Sanmarco MF, Azen SP, Cashin-Hemphill L: Beneficial effects of combined colestipol-niacin therapy on coronary atherosclerosis and coronary venous bypass grafts. JAMA 1987;257:3233–3240.
3. Hjermann I, Velve Byre K, Holme I, Leren P: Effect of diet and smoking intervention on the incidence of coronary heart disease: report from the Oslo Study Group of a randomized trial in healthy men. Lancet 1981; 2:1303–1310.
4. Arntzenius AC, Kromhout D, Barth JD, Reiber JHC, Bruschke AVG, Buis B, van Gent CM, Kempen-Voogd N, Strikwerda S, vander Velde EA: Diet, lipoproteins, and the progression of coronary atherosclerosis: the Leiden Intervention Trial. N Engl J Med 1985; 312:805–811.
5. Expert Panel: Report of the National Cholesterol Education Program Expert Panel on detection, evaluation, and treatment of high blood cholesterol in adults. Arch Intern Med 1988; 148:36–69.
6. National Heart, Lung, and Blood Institute. The Lipid Research Clinic Population Studies Data Book. Bethesda, MD: National Heart, Lung, and Blood Institute, Lipid Metabolism Branch, pub. no. 80–1537, vol 1, 1980.
7. Rastam L, Luepker RV, Mittelmark MB, Murray KM, Slater JS, Blackburn H: Population screening and referral for hypercholesterolemia. Am J Prev Med 1988; 4:249–254.
8. Rastam L, Luepker RV, Pirie PL: Effect of screening and referral on follow-up and treatment of high blood cholesterol levels. Am J Prev Med 1988; 4:244–248.
9. Block L, Banspach SW, Gans K, Harris C, Lasater TM, Lefebvre C, Carleton RA: Impact of public education and continuing medical education on physician attitudes and behavior concerning cholesterol. Am J Prev Med 1988; 4:255–260.
10. Manninen V, Elo O, Frick H, Haapa K, *et al:* Lipid alterations and decline in the incidence of coronary heart disease in the Helsinki Heart Study. JAMA 1988; 260:641–651.
11. Wilson SW, Hulley SB, Burrows ML, *et al:* Serum lipid and lipoprotein responses to the American Heart Association fat-controlled diet. Am J Med 1977; 51:491–450.
12. National Center for Health Statistics: Plan and operation of the Second National Health and Nutrition Examination Survey 1976–1980. Hyattsville, MD: National Center for Health Statistics, DHHS Pub No. (PHS) 81–1317, July 1981.
13. Forthofer RN: Investigation of nonresponse bias in NHANES II. Am J Epidemiol 1983; 117:507–515.
14. US Department of Agriculture, Agriculture Research Service: Composition of Foods: Raw, Processed, Prepared. Agriculture Handbook No 8, 1968, Expansion (March 1972). Washington, DC: Govt Printing Office, 1972.
15. National Institutes of Health: The dietary management of hyperlipoproteinemia. A handbook for physicians and dietitians. DHEW Pub. No. (NIH) 78–110. Bethesda, MD: National Heart, Lung, and Blood Institute, National Institutes of Health, 1978.
16. Levy RI (moderator): National Institutes of Health Consensus Conference: Dietary and drug treatment of primary hyperlipoproteinemia. Ann Intern Med 1972; 77:267–294.
17. Levy RI, Bonnell M, Ernest ND: Dietary management of hyperlipoproteinemia. J Am Dietet Assoc 1971; 58:406.
18. Leren P: The Oslo diet-heart study. Eleven-year report. Circulation 1960; 62:935–942.
19. Brown HB: Food patterns that lower blood lipids in man. J Am Dietet Assoc 1971; 58:303–311.
20. La Rosa J: Hyperlipoproteinemia; Dietary Management. Postgrad Med 1972; 52:75–79.
21. Dietary Goals for the United States, 2nd Ed. Washington, DC: US Senate, Select Committee on Nutrition and Human Needs, December 1977.
22. Am J Clin Nutr Symposium Report on Dietary Factors Relating to the Nation's Health. Am J Clin Nutri 1979; 32:126–142.
23. Report of Inter-Society Commission for Heart Disease Resources. Primary Prevention of the Atherosclerotic Diseases. Circulation 1970; 42:A80–95.
24. Hidiroglou MA, Fuller WA, Hickman RD: SUPERCARP, Survey Section, Statistical Laboratory, Iowa State University, Sixth Ed, October 1980.
25. Draper NR, Smith H: Applied Regression Analysis. New York: John Wiley & Sons, 1966.
26. Cook RD: Detection of influential observations in linear regression. Technometrics 1977; 19:15–18.
27. Grundy SM: Cholesterol and coronary artery disease: a new era. JAMA 1986; 256:2849–2858.
28. Schucker B, Wittes JT, Cutler JA, Bailey K, Mackintosh DR, Gordon DJ, Haines CM, Mattson ME, Goor RS, Rifkind BM: Change in physician perspective on cholesterol and heart disease. JAMA 1987;258:3521–3526.
29. Block G: A review of validations of dietary assessment methods. Am J Epidemiol 1982; 115:492–505.
30. Subcommittee on Criteria for Dietary Evaluation, National Research Council: Nutrient Adequacy: Assessment Using Food Consumption Surveys. Washington, DC: National Academy Press, 1986.
31. Mann HI, Marr JW: Coronary heart disease prevention trials of diet to control hyperlipidemia. *In:* Miller NE, Lewis B (eds): Lipoproteins, Atherosclerosis, and Coronary Heart Disease. Amsterdam: Elsevier/North-Holland Biomedical Press, 1981; 197.
32. National Diet-Heart Study Research Group. The National Diet-Heart Study final report. Circulation 1968; (Suppl 1).
33. Whyte HM, Havenstein N: A perspective view of dieting to lower the blood cholesterol. Am J Clin Nutr 1976; 29:784–790.
34. Dougherty RM, Fong AKH, Iacono JM: Nutrient content of the diet when the fat is reduced. Am J Clin Nutr 1988; 48:970–979.
35. Wrisley DW, Rubenfire M: Ineffectiveness of standardized dietary counseling in hypercholesterolemic patients with coronary disease. J Cardiopul Rehab 1988; 8:226–230.
36. Sivarajan ES, Almes MJ, Mansfield LW: Limited effects of outpatient teaching and counseling after myocardial infarction: A controlled study. Heart Lung 1983; 12:65–73.

31

Spanish Version of the Nottingham Health Profile: Translation and Preliminary Validity

JORDI ALONSO, MD, JOSEP MARIA ANTO, MD, AND CONCHI MORENO, MD, MPH

Abstract: We report the transfer into Spanish of a multidimensional measure of perceived health originally developed in Great Britain, the Nottingham Health Profile (NHP), and an assessment of the preliminary validity of the version is presented.

Translation of the questionnaire was obtained from experts and from a Spanish monolingual lay group. Construct validity of the version was assessed in two studies: testing relationship of NHP scores to other self-reported measures of health in a general population survey; and comparing NHP scores for a group of frequent users and for a group of non-users of primary health services.

Mean scores of NHP dimensions were higher for people with poorer self-reported health and higher for the frequent health services users than for the non-users.

Findings suggest that the Spanish version of the NHP is culturally equivalent to the original questionnaire, and has a similar level of construct validity. Nevertheless, further research on reliability and on the weighting system is required to establish the equivalence of the Spanish version definitively. (*Am J Public Health* 1990; 80:704–708.)

Introduction

The impetus to produce reliable and valid measures of health has grown dramatically as the need for better evaluation of health services has increased. In the last two decades a number of questionnaires have been designed which measure the physical, mental, and social aspects of health.[1] Although most of these measures have been developed and tested in English-speaking cultures, mainly in the United States, they are widely used in different countries and in non-English-speaking cultures.

The content of health status measures as well as the wording and the form of administration are very dependent on cultural values.[2,3] Transferring a validated instrument to a different culture is an efficient strategy, since developing a new measure is a complex and time-consuming task, requiring much expertise. However, this process should follow a methodology that ensures a similar level of validity, reliability, and cultural meaning for the new version.[4,5] This methodology includes several steps: translation (ensuring conceptual rather than linguistic equivalence), and testing for feasibility, validity, and reliability. Furthermore, measures containing scaled items usually include specific weights for each item, thus making it necessary to validate the weights for the adapted version as well, in order to assure that scores obtained in the original and adapted version have similar meaning.[6,7]

In past work[8] we identified the need for a measure to adequately monitor health status of the population and equity in health services utilization. We have focused our work on adapting a validated measure rather than on developing a new one. We selected the Nottingham Health Profile (NHP), a validated measure of perceived distress developed in Great Britain,[9–12] and previously adapted in Sweden[7] and France.[13]

The NHP is a self-administered questionnaire containing a first part with 38 statements belonging to six dimensions of health (Energy, Pain, Emotional Reactions, Sleep, Social Isolation, and Physical Mobility). The respondent answers "yes" if the statement adequately reflects his/her current status or feeling, or "no," otherwise.[9] British researchers weighted each item in a dimension using Thurstone's paired comparisons method.[14] Part II contains questions on seven areas of daily life.

For transferring the NHP into Spanish, the following strategy was designed: first, a conceptually equivalent translation; second, a preliminary assessment of the validity of the version obtained; and finally develop Spanish weights, contingent upon the establishment of the validity of the unweighted version.

Methods

Translation Process

Ideally, a panel of bilingual people should be used to obtain translations and blind back-translations.[15] Because it was difficult to find a satisfactorily representative panel of bilinguals (same fluency in Spanish as in English, middle/lower socioeconomic class, and experiencing chronic illnesses), a specific methodology for adapting the NHP into Spanish was designed by one of the NHP authors (S. Hunt).[16] After complete explanations by Dr. Hunt, translations of each item were obtained by two of us (JMA and JA). These translations were back-translated into English and discussed again. A list containing translated items and alternative translations was made. Close agreement was reached in most items and they were graded as A "satisfactory agreement" (32 out of 45, 71 percent), B "almost satisfactory but one or two words uncertain" (nine items, 20 percent), and C "doubtful translation" (four items, 9 percent).

In order to overcome these disagreements, a group of 10 Spanish unskilled workers listened to anecdotes that reflected all the items on the questionnaire and described them freely in their own words. These words were added to the list of alternative translations. This list was later presented to the same group in order to point out the expressions they found odd and to select those that would conform to their language usage. The final version included the most popular expressions for items B and C (see Appendix). A pre-test of this version was carried out with a group of cardiology and rheumatology hospital outpatients who were asked to complete the questionnaire and to identify the statements difficult to understand, those that were acceptable, and those easily understood.

From the Department d'Epidemiologia i Salut Pública, Institut Municipal d'Investigació Mèdica (IMIM), Ajuntament de Barcelona, Spain. Address reprint requests to Jordi Alonso-Caballero, MD, Department d'Epidemiologia i Salut Pública, IMIM, Passeig Marítim, 25-29, 08003 Barcelona, Spain. This paper, submitted to the Journal April 27, 1989, was revised and accepted for publication October 4, 1989.

Having obtained a Spanish version of the NHP, a translation into Catalan (language of romanic origin similar to Spanish, spoken by some eight million people mainly in the northeastern part of Spain) was also made. Seven Catalan-Spanish bilinguals, who were easy to convene, were used to obtain translations and back-translations and also to decide the exact final wording. The close agreement between both Catalan and Spanish versions led us to include both versions in further studies of the Spanish NHP.

Validation Studies

Achieving conceptual and linguistic equivalence when transferring a questionnaire from one culture to another does not entirely guarantee that the validity of the original version stands in the new culture. Although the method used to translate the NHP into Spanish followed prescribed procedures, the results of this translation were further tested to determine whether it performed in a manner similar to the British NHP.

In order to make an assessment of the validity of the Spanish version of the NHP, construct validity was tested in two studies. First, the relationship of NHP scores to other self-reported measures of health was tested; second, NHP scores for a group of frequent users and for a group of non-users of primary health services were compared.

In the first study, 1,220 participants ages 41 years and over completed the NHP (75 percent Spanish version and 25 percent Catalan version) as part of a random sample health survey of the city of Barcelona.[17] They also provided information about self-rated health (SRH), chronic conditions selected from a list of 34 conditions, chronic limitation of activity due to health problems, and restricted activity in the two weeks before the interview. These variables had been used in previous studies in Spain.[18,19] Social class was assigned to the respondents based on the occupation of the head of the household, following an adaptation[20] of the British Registrar's Social Classification.[21]

In the second study, clinical records of individuals ages 20 and over were sampled systematically from the registry of the Health Center of Barañain (Navarra). Following the criteria of a previous validity study of the British NHP,[11] people were classified as "non-users" if no medical visits were registered in the last six months, and as "frequent users" if three or more visits were made by individuals less than 50 years old, or five or more visits for individuals age 50 and over. As illness was the construct of interest, routine medical examinations were not included. In total, 396 people were considered eligible for the study (195 "non-users" and 201 "frequent users"). They were asked to complete the Spanish version of the NHP and to give information on self-rated health and chronic conditions. Complete responses were obtained from 338 individuals (85.3 percent of the eligible). Following the previous British study,[11] influence of age and sex in the analysis was eliminated by randomly selecting from the respondents a subsample of 276 people containing equal numbers of "non-users" and "frequent users" in each age and sex group. Four age ranges were categorized by 10-year intervals (20–29, 30–39, 40–49, and 50 and over).

We calculated scores for each NHP dimension in these studies by adding all the answers to the items in the dimension, giving the value of 0 for answers of "no" and 1 for answers of "yes". To obtain a final score in each dimension, and to overcome the fact of having a different number of items in some of the dimensions, each sum was multiplied by 100 and divided by the number of items in the dimension. Possible scores ranged from 0 (all answers of "no" in the dimension, denoting absence of distress) to 100 (all answers of "yes", denoting maximal distress). Statistical significance was assessed using nonparametric tests (one-tailed Wilcoxon-Mann-Whitney, and Kruskal Wallis tests at a 5 percent level of significance[22]) because NHP responses do not follow a normal distribution.[9]

Mean scores of the NHP for the general population ages 41 and over of the city of Barcelona are presented in Table 1. Highest mean scores corresponded to Sleep and to Emotional Reactions (23.2 and 20.8, respectively), while the lowest was that of Social Isolation (7.2). Women scored considerably higher than men in all the dimensions. Mean scores were higher for social classes IV–V (manual workers) than for social classes I–II (directors, professionals and others).

People rating their health as "very good" scored 2.9 in Energy, 3.7 in Pain, and 4.9 in Social Isolation, as compared with those with "poor" or "very poor" health, scoring 64.3, 58.0, and 21.4, respectively (Table 2). People with at least one of the 34 chronic conditions listed scored significantly higher than those not reporting any condition, the score being higher as the number of conditions increased. Similar differences in

TABLE 1—Mean Scores for each Dimension of the Spanish Version of the NHP in the General Population Ages 41 and over of Barcelona.

	Energy	Pain	Emotional Reactions	Sleep	Social Isolation	Mobility (n)
Total	14.1	15.6	20.8	23.2	7.2	14.9 (1,220)
Sex						
Male	8.5	9.1	16.1	17.6	5.7	10.6 (578)
Female	19.0	21.5	25.0	28.2	8.6	18.8 (642)
Age group						
41–49	8.9	10.7	19.8	16.7	4.4	9.0 (320)
50–59	12.6	14.4	20.9	20.7	7.0	11.5 (405)
60–69	15.4	18.4	18.7	27.1	7.8	17.5 (292)
70+	23.3	21.8	25.0	32.8	11.5	27.3 (203)
Social Class (age-adjusted scores)*						
I–II	10.3	8.0	15.7	16.2	4.3	11.2 (256)
III	10.5	11.5	15.1	18.0	5.5	12.9 (299)
IV–V	17.0	20.8	25.5	27.1	8.9	17.4 (606)

*59 individuals not classified.

TABLE 2—Mean Scores for Each Dimension of the Spanish Version of the NHP by Level of Self-reported Health Variables (convergent validity variables), General Population Ages 41 and over of Barcelona

Characteristics	Energy	Pain	Emotional Reactions	Sleep	Social Isolation	Mobility (n)
Self-Rated Health*						
Very good	2.9	3.7	12.1	12.0	4.9	7.0 (119)
Good	6.1	7.8	14.8	17.2	4.9	9.0 (692)
Fair	25.5	28.1	30.6	34.2	10.6	23.8 (350)
Poor or very poor	64.3	58.0	47.4	56.1	21.4	49.3 (59)
Chronic Conditions**						
No	3.4	3.5	11.4	10.6	2.6	4.3 (245)
Yes	16.8	16.7	23.2	26.4	8.4	17.7 (975)
Number of Chronic Conditions*						
0	3.4	3.5	11.4	10.6	2.6	4.3 (245)
1	5.5	7.4	16.8	17.4	5.7	9.3 (278)
2	12.3	13.9	19.6	24.0	7.5	13.9 (237)
3	18.1	21.6	24.3	23.3	8.3	18.9 (161)
4	19.0	24.0	26.9	31.6	9.2	21.5 (129)
5+	38.5	37.2	35.0	43.3	14.3	32.1 (170)
Chronic Limitation of Activity**						
No	10.7	12.5	19.0	21.0	6.6	12.5 (1,086)
Yes	42.0	41.0	35.0	42.1	12.6	35.1 (134)
Restricted Activity Days (in the past two weeks)**						
No	11.9	13.8	19.7	22.1	6.8	13.4 (1,133)
Yes	41.8	39.6	33.5	37.3	12.3	33.4 (87)

*Kruskal-Wallis test: $p < 0.0001$, for all dimensions.
**Wilcoxon-Mann-Whitney one-tailed test: $p < 0.0001$, for all dimensions.

size and direction were found for chronic limitation of activity and for restricted activity days (Table 2). Logistic regression analysis showed that NHP scores remained significantly associated with the construct validity variables, when age, sex, and social class were taken into account.

As shown in Table 3, the mean scores in each of the NHP dimensions were higher for the "frequent users" than for the "non-users." The most important relative differences corresponded to Pain and Energy dimensions (50 percent or more increase). Also, people reporting better health scored lower than those reporting worse health. NHP scores showed an ascending gradient related to reported number of chronic conditions (Table 3).

Discussion

Accurate translation is a first requirement in the process of transferring a health measure from the culture of origin to another culture.[2,5,15,23] However, it is a common practice that researchers themselves directly translate health questionnaires using technical and/or uncommon expressions which can affect validity, reliability, and comparability of results.

The translation methodology used for transferring the NHP into Spanish ensured a conceptually equivalent version.[24] The high level of agreement achieved in most back-translated items supports this equivalence. The participation of the monolingual lay panel in the translation ensured that statements of the new version were based on lay

TABLE 3—Mean Scores for each Dimension of the Spanish Version of the NHP by "Non-users" and "Frequent Users" and by other Convergent Validity Variables: Study of the Health Center of Barañain (Pamplona) (n = 276)

Characteristics	Energy	Pain	Emotional Reactions	Sleep	Social Isolation	Mobility (n)
Use of Health Services**						
Non-users	8.7	8.3	16.9	16.7	6.4	10.2 (138)
Frequent users	13.0	13.8	23.9	23.8	8.8	13.8 (138)
Self-Rated Health*						
Very good	0.0	2.1	7.7	7.8	2.1	3.6 (38)
Good	4.5	6.6	15.4	14.1	6.2	7.1 (158)
Fair	25.1	21.9	35.2	39.1	10.3	25.0 (69)
Poor or very poor	57.6	37.5	50.6	24.0	30.9	33.7 (11)
Number of Chronic Conditions*						
0	5.3	1.0	10.2	8.4	3.1	3.9 (38
1	7.1	5.8	12.8	13.8	3.1	8.2 (66
2	10.8	11.2	20.8	17.4	8.2	11.5 (78)
3	12.6	11.3	27.3	21.4	13.6	15.6 (45)
4	14.1	25.0	28.5	36.4	11.5	20.6 (26)
5+	24.4	27.2	35.5	52.4	10.0	25.6 (23)

*Kruskal-Wallis test: $p < 0.01$, for all dimensions.
**Wilcoxon-Mann-Whitney one-tailed test: $p < 0.05$, for all dimensions.

expressions of health perceptions, as the original instrument. The fact that similar expressions had been obtained for other European versions of the NHP, further suggests that NHP items address aspects of health perceptions that have conceptual equivalence for these cultures.[7,13]

As mentioned above, the strategy for transferring the NHP into Spanish included the assessment of validity of the version before obtaining the weights of the items. As there is no gold standard against which validity of complex health status measures could be evaluated,[1,25,26] construct validity rather than criterion validity of the Spanish version has been assessed.

The results of the studies presented preliminarily suggest that the unweighted Spanish version of the NHP has construct validity. Mean scores in every NHP dimension are clearly different for individuals with diverse degrees of perceived health as assessed by a variety of other construct validity variables. All differences were in the expected direction: the lower the level of the perceived health indicator the higher the NHP score. The similarity of the results from both studies, with different designs and populations, further supports the validity of the questionnaire.

A similar distribution of NHP scores across different self-rated health (SRH) categories has been observed in several studies in Britain.[9,27,28] SRH has been shown to be a good predictor of health outcomes, even after adjusting for a number of variables including "objective" health status.[29,30] The consistency and the strength of the relationship between NHP scores and SRH is evidence of the validity of the questionnaire. In both studies presented, an increase was observed in mean NHP scores as the number of chronic conditions increased. Even though the severity of the conditions included was not considered, and the fact that conditions reported may reflect both medically diagnosed and patient perceived health problems,[31–33] it was expected that, on the average, people reporting more health conditions would have a worse perception of their health.

The approach of the first study for assessing construct validity, i.e., to compare scores for the new measure for different levels of self-reported health variables, has been previously utilized.[26,34,35] Most of these studies have reported high correlations among the health measures and the construct validity variables, similar to the results presented for the Spanish NHP. However, the relationship found between NHP responses and construct validity variables may be overestimated as responses to both were obtained directly by the respondent.

Conversely, in the second study, the number of visits was obtained from clinical records, independently of the measure of the responses to NHP. Thus, mean score differences between non-users and frequent users provide further evidence of the validity of the Spanish version of the NHP. However, these differences, although statistically significant, are not very large. This seems to be due to the fact that the scores of the "frequent users" are not so high as might be expected. The most likely explanation of this could be that people in the poorest health were misclassified due to a reduced likelihood of home visits by the doctor being recorded. Another explanation can be found in the fact that many of the "non-users" are non-symptom-free. A comparison of the distribution of health problems suggests that highest differences between these two groups were for conditions which have low potential impact on NHP scores (e.g., hypertension and hearing problems).

Nevertheless, to completely assess the equivalence of the questionnaire, similarity of the weights of the items should be established, as it has been done for other NHP European versions[7,13] and for the British version of the Sickness Impact Profile.[6] As mentioned above, the British authors of the NHP developed weights for the items in each dimension, using Thurstone's method of paired comparison;[14] weights being calculated by considering the proportion of times each statement was judged to be more severe than each of the other statements in the dimension by a representative group of lay people.

Developing the weights for the Spanish version would provide an important basis for definitively assessing validity of the questionnaire and for ensuring cross-cultural comparability.

APPENDIX

English and Spanish Versions of Selected Items of the Nottingham Health Profile (and graded agreement of back-translation)

British Item	Spanish Item	(Agreement)
Energy		
The days seem to drag	Los días se me hacen interminables	(A)
I'm tired all the time	Siempre estoy fatigado	(C)
Pain		
I have pain at night	Tengo dolor por las noches	(A)
Emotional Reactions		
Worry is keeping me awake at night	Las preocupaciones me desvelan por la noche	(A)
I lose my temper easily these days	Ultimamente me enfado con facilidad	(B)
Things are getting me down	Las cosas me deprimen	(C)
Sleep		
I take tablets to help me sleep	Tomo pastillas para dormir	(A)
I'm waking in the early hours of the morning	Me despierto antes de hora	(C)
Social Isolation		
I'm finding it hard to get on with people	Me cuesta llevarme bien con la gente	(A)
I'm finding it hard to make contact with people	Ultimamente me resulta difícil contactar con la gente	(C)
Physical Mobility		
I can only walk about indoors	Sólo puedo andar por dentro de casa	(A)
I find it hard to reach for things	Me cuesta coger las cosas	(C)

(A) Satisfactory translation
(B) One or two words uncertain
(C) Doubtful translation

ACKNOWLEDGMENTS

The authors want to acknowledge Dr. Sonja Hunt for her mastership and personal help. They also thank Dr. Marilyn Bergner, PhD for her support and scientific advice and Dr. Encarna Grifell for her help during data collection and management. Parts of this paper have been presented at the 1988 and the 1989 Scientific Meetings of the Spanish Epidemiological Association (San Sebastián, 1988 and Santiago de Compostela, 1989).

The work has been partially supported by the following grants: CAICYT (PA85-0240-CO3-02), "Area de Salut Pública de l'Ajuntament de Barcelona, 1986" and "Proyectos de Investigación Sanitaria y de Servicios Sociales del Gobierno de Navarra, 1986."

REFERENCES

1. McDowell I, Newell C: Measuring Health. A guide for rating scales and questionnaires. Oxford: Oxford University Press, 1987.
2. Hunt SM: Cross-cultural issues in the use of socio-medical indicators. Health Policy 1986; 6:149–158.

3. Lonner WJ, Berry JW (eds): Field Methods in Cross-Cultural Research. Beverly Hills: Sage Publications, 1986.
4. Patrick DL: Standardization of comparative health status measures: Using scales developed in America in an English-speaking country. *In:* Survey Research Methods: Third Biennial Conference. Hyattsville, MD: US Dept of Health and Human Services, Pub. No. (PHS) 81-3268, 1981; 216–220.
5. Deyo RA: Pitfalls in measuring the health status of Mexican Americans: Comparative validity of the English and Spanish Sickness Impact Profile. Am J Public Health 1984; 74:569–573.
6. Patrick DL, Sittampalam Y, Somerville SM, Carter WM, Bergner M: A cross-cultural comparison of health status values. Am J Public Health 1985; 75:1402–1407.
7. Hunt SM, Wiklund I: Cross-cultural variation in the weighting of health statements: A comparison of English and Swedish valuations. Health Policy 1987; 8:227–235.
8. Alonso J, Antó JM: Desigualtats de salut a Barcelona. Gac Sanit 1988; 2(4):4–12.
9. Hunt SM, McEwen J, McKenna SP: Measuring Health Status. London: Croom Helm, 1986.
10. Hunt SM, McKenna SP, McEwen J, Backett EM, Williams J, Papp E: A quantitative approach to perceived health status: A validation study. J Epidemiol Community Health 1980; 34:281–286.
11. Hunt SM, McKenna SP, McEwen J, Williams J, Papp E: The Nottingham Health Profile: Subjective health status and medical consultations. Soc Sci Med 1981; 15A:221–229.
12. McKenna SP, Hunt SM, McEwen J, Pope C: Changes in the perceived health of patients recovering from fractures. Public Health 1984; 98:97–102.
13. Bucquet D, Condon S: Adaptation into French of the Nottingham Health Profile and operational characteristics of the French version. Villejuif 94807 CEDEX: INSERM (Unité 164), 1988.
14. McKenna SP, Hunt SM, McEwen J: Weighting the seriousness of perceived health problems using Thurstone's method of paired comparisons. Int J Epidemiol 1981; 10:93–97.
15. Brislin RW: The wording and translation of research instruments. *In:* Lonner WJ, Berry JW (eds): Field Methods in Cross-cultural Research. Beverly Hills: Sage Publications, 1986; 137–164.
16. Antó JM, Alonso J: A health profile for use in Spain. (letter) Am J Public Health 1986; 76:711.
17. Alonso J, Antó JM: Enquesta de Salut de Barcelona 1986. Barcelona: Ajuntament de Barcelona, 1989.
18. Antó JM, Domingo A, Companys A: Encuesta de salud de Barcelona. Barcelona: Area de Salud Pública del Ayuntamiento de Barcelona, 1985.
19. Departamento de Sanidad y Consumo: Encuesta de Salud de la Comunidad Autónoma Vasca. Vitoria: Servicio central de publicaciones del Gobierno Vasco, 1987.
20. Domingo A, Marcos J: Propuesta de un indicador de "clase social" basado en la ocupación. Gac Sanit 1989; 3(10):320–326.
21. Registrar General: Classification of Occupations. London: HMSO, 1970.
22. Siegel S, Castellan NJ: Nonparametric statistics for the behavioral sciences. 2nd Ed. New York: McGraw-Hill, 1988; 128–144, 206–216.
23. Gilson B, Erickson D, Chavez CT, Bobbit RA, Bergner M, Carter WB: A chicano version of the Sickness Impact Profile (SIP). Cult Med Psychiatry 1980; 4:137–150.
24. Bice TW, Kalimo E: Comparisons of health-related attitudes: A cross-national, factor analytic study. Soc Sci Med 1971; 5:283–318.
25. Kaplan RM, Bush JW, Berry CC: Health status: Types of validity and the Index of Well-being. Health Serv Res 1976; 4:478–507.
26. American Psychological Association: Standards for educational and psychological tests. Washington, DC: American Psychological Association, 1974.
27. Leavey R, Wilkin D: A comparison of two survey measures of health status. Soc Sci Med 1988; 27:269–275.
28. Hunt SM, McEwen J, McKenna SP: Social inequalities and perceived health. Effective Health Care 1985; 4:151–160.
29. Mossey JM, Shapiro E: Self-rated health: a predictor of mortality among the elderly. Am J Public Health 1982; 72:800–808.
30. Kaplan GA, Camacho T: Perceived health and mortality: A nine-year follow-up of the Human Population Laboratory Cohort. Am J Epidemiol 1983; 117:291–304.
31. Kaplan SH: Patient reports of health status as predictors of physiologic health measures in chronic disease. J Chronic Dis 1987; 40(suppl):27s–35s.
32. Feldman J: The household interview survey as a technique for the collection of morbidity data. J Chronic Dis 1960; 11:535–537.
33. Kroeger A: Response errors and other problems of health interview surveys in developing countries. World Health Stat Q 1985; 1:15–37.
34. Bergner M, Rothman ML: Health status measures: An overview and guide for selection. Ann Rev Public Health 1987; 8:191–210.
35. Rea JL, Quinn RJ, Hoeffer MA: Measuring overall health: An evaluation of three important approaches. J Chronic Dis 1987; 40(suppl):7s–21s.

New NCHS Report on Testing Fitness and Physical Activity

Assessing Physical Fitness and Physical Activity in Population-Based Surveys is a new report from the National Center for Health Statistics. The publication is a comprehensive review of the state-of-the-art and future directions in testing fitness and measuring physical activity.

The objectives set for the nation to improve health status through disease prevention and health promotion depend, in part, upon changing patterns of physical fitness and increasing levels of physical activity in the population. This reference volume is a comprehensive review of many topics associated with fitness testing and measurement of physical activity, including:

- Design issues and alternatives in assessing physical fitness among healthy adults as well as special populations;
- Assessment of energy turnover, energy intake or energy expenditures;
- Fitness and activity assessment of special populations of children, adolescents, women, and older adults;
- Evaluation of the health effects of demanding work on and off the job;
- Effects of physical activity and fitness on health; and
- Evaluation of the interrelationships among physical fitness and activity assessments and the epidemiologic uses of general population assessments of physical activity patterns and physical fitness.

Assessing Physical Fitness and Physical Activity in Population-Based Surveys can be purchased for $31; stock no. 017-022-010224, from the Superintendent of Documents, Government Printing Office, Washington, DC 20402.

2T

Public Health Briefs

Evaluating Community-based Nutrition Programs: Assessing the Reliability of a Survey of Grocery Store Product Displays

Allen Cheadle, PhD, Bruce Psaty, MD, PhD, Edward Wagner, MD, MPH, Paula Diehr, PhD,
Thomas Koepsell, MD, MPH, Susan Curry, PhD, and Michael Von Korff, PhD

Abstract: A pilot test of a survey of grocery store product displays was conducted to measure the amount of health-education information provided and the proportion of the display devoted to "healthier" products. Inter-rater reliability ranged between 0.73 and 0.78 for the healthiness indices and between 0.30 and 0.67 for the health education measures. Test-retest reliability ranged from 0.44 to 1.0. (*Am J Public Health* 1990; 80:709–711.)

Introduction

There have been a number of recent efforts directed at entire communities to modify dietary behavior in order to reduce the risk of cardiovascular disease and cancer.[1–4] Evaluation of these interventions requires "community-level indicators" of program effectiveness—measures of the community environment that are not merely aggregates of individual behavior.[5] We measured the reproducibility of a survey of grocery store product displays to be used as part of the evaluation of community-based nutrition programs. Such surveys are particularly useful when grocery stores and supermarkets play a role in the intervention, as in the Giant Foods project[1] and in the Henry J. Kaiser Family Foundation Community Health Promotion Grants Program (CHPGP).*

Methods

We surveyed four general product areas within each grocery store: fresh produce, meat, milk, and bread. For each major product area, two types of information were recorded: presence or absence of health-promotion items, and the physical dimensions of product displays. The protocol took 30–45 minutes for an experienced rater to complete.

- *Health-promotion items* were defined as health education activities (usually printed material) on or near the displays, which provided nutritional information or increased awareness of healthy food choices, and which were independent of product packaging. Examples of health-promotion items included: shelf labeling, posters, recipes, and store health-information centers.
- *The measurements of the displays* were designed to create an index of the relative amount of space occupied by "healthy" products in each product area, calculated as the proportion of shelf space devoted to such products (e.g., those low in fat) relative to the overall size of the display of similar foods.

The sampling frame consisted of full-service supermarkets, defined as grocery stores with two or more check-out stands, carrying fresh produce and fresh meat, that were listed in the Yellow Pages telephone directory. The sample for the Seattle pilot test of inter-rater reliability included 13 independent stores and 24 chain stores. For this part of the field test, each store was surveyed by two raters who visited the four product areas in a different sequence. Test-retest reliability was evaluated on an additional random sample of 61 stores in five communities that are part of the overall Kaiser Foundation evaluation. These test-retests were carried out by the same rater on some occasions and by different raters on others, with a variety of time intervals separating the visits.

Three indices of display "healthfulness" are presented here: the proportion of poultry and fish in the meat display, the proportion of reduced-fat milk in the milk display, and the proportion of 100% whole wheat bread in the bread display. Health promotion items were coded simply as present or absent for each of the major product areas. To assess reliability, we used the intra-class correlation coefficient[6] for the continuous variables and weighted kappas[7] for the discrete variables.

Results

As Table 1 shows, chain stores (particularly national or regional chains) more often offered health promotion items than did independent stores. By contrast, there was little apparent difference in the three display variables by store type. Table 2, column (1) gives inter-rater reliability coefficients computed for the first visits to the 37 stores in the pilot sample. Reliability for the display variables was fairly high, in the range of .7 to .8, but was lower for the health promotion items. The low figures for the milk and meat health promotion items were partly due to an open-ended format for recording these items in the pilot survey; a closed format used in the later surveys produced better results. The figures in column

---

From the University of Washington, Seattle, Departments of Health Services (Cheadle, Wagner, Diehr, Koepsell, Curry, Von Korff); Biostatistics (Diehr); Medicine (Psaty); Epidemiology (Psaty, Koepsell); and the Center for Health Studies, Group Health Cooperative of Puget Sound (Wagner, Curry, Von Korff). Address reprint requests to Allen D. Cheadle, PhD, Department of Health Services, JD-43, School of Public Health and Community Medicine, University of Washington, Seattle, WA 98195. This paper, submitted to the Journal March 2, 1989, was revised and accepted for publication November 27, 1989.

*Wagner EH, Kehrer B: The Henry J. Kaiser Family Foundation Community Health Promotion Program: Evaluation design considerations. Presented at the annual meeting of the International Epidemiological Association, Helsinki, Finland, August 6–13, 1987.

TABLE 1—Means of Grocery Store Survey Variables, by Store Ownership Category, Seattle Pilot Test Stores

Variables	Independent (n = 13)	Chains Local (n = 13)	 National/Regional (n = 12)	 All (n = 37)
Health promotion items[a]				
Produce display	46.1	50.0	100.0	64.9
Meat display	69.2	100.0	100.0	89.2
Milk display	30.8	41.7	50.0	40.5
Display variables[b]				
% Poultry + fish	44.4 (4.9)	50.6 (6.9)	43.8 (5.8)	46.2 (6.5)
% Reduced-fat milk	67.9 (8.9)	66.6 (6.4)	62.5 (9.8)	65.7 (8.6)
% Whole-wheat bread	5.9 (2.7)	5.8 (2.1)	4.4 (1.2)	5.4 (2.1)

NOTES: Standard deviation in parentheses.

[a] Percent of stores with health education materials in the indicated section. Store was counted as having health promotion items if either of the two raters recorded them.

[b] Percent of the display with indicated products. Measurements for each store are the average of the two raters. Definitions for numerator and denominator were as follows:

Meat: Shelf space occupied by poultry and fish/Total
Total = Fresh meat only; excludes frozen, canned, smoked or otherwise processed meat.
Milk: Shelf space occupied by 2% fat and skim milk/Total
Total = 2% fat, skim and whole milk only (half-gallon cartons)
Bread: Shelf space occupied by 100% whole wheat bread/Total
Total = Sliced, packaged loaves; excludes specialty bakery section, desert pastry, french bread, baguettes.

TABLE 2—Reliability Coefficients for Display and Health Promotion Item Measurements*

Variables	Different raters, same occasion[a]	Different raters, different occasions[b]	Same rater, different occasions[c]	Same rater, same occasion[d]
	(n = 37)	(n = 15)	(n = 27)	(n = 19)
Health promotion items				
Produce display	.67	1.00	.92	—
Meat display	.36	.63	.91	—
Milk display	.30	.46	1.00	—
Display Variables				
% Poultry + fish	.78	.86	.44	.98
% Reduced-fat milk	.75	.68	.58	.91
% Whole-wheat bread	.73	.47	.65	.93

*Statistical methods:
Display Variables: Intra-class correlation coefficients
Health Promotion Items: Weighted kappas

[a] Site: Seattle, WA (n = 37 stores)
[b] Site: Solano County, CA (n = 15)
[c] Sites: San Francisco, CA (n = 15), Orange County, CA (n = 12)
[d] Sites; Santa Cruz, CA (n = 15), Fresno, CA (n = 4)

(2), Table 2, show the correlations between two raters over a one-week time interval. This should approximate contemporaneous inter-rater reliability measures since few changes in health education materials were likely to have been made in this brief period. The revised format raised inter-rater reliability for the meat and milk displays—to 0.63 to 0.46, respectively.

The intra-class correlation coefficients presented in column (4), Table 2, all above 0.9, indicate high reliability in the same rater measuring the same display twice. Thus, most of the variation between occasions by the same rater was due to changes made by stores. The relatively high coefficients for health promotion items in column (3) suggest that these changed infrequently for the time intervals represented here. Display items showed lower intra-class correlation coefficients over time, particularly the meat display.

Discussion

Although sales data would reflect consumption most accurately, these are often unavailable to researchers. In the alternative measures which we used, measurements were made unobtrusively, and all of the stores approached for the pilot test agreed to participate; the response rate for the main study was 88 percent. Inter-rater reliability in the pilot was relatively high, particularly for the display variables. The lower inter-rater reliability figures for the health promotion items may have been due to the one-week interval between rater visits although, as noted earlier, significant changes were unlikely to have been made in that brief period. Test-retest reliability results indicated that both health promotion and display measures were relatively stable over periods ranging from one week to two months. Validity of the approach is currently being assessed, first by comparing the grocery store measures to the socioeconomic characteristics of people in the surrounding area, and second by correlating the survey measures with responses to dietary surveys of randomly sampled individuals living near the stores.

ACKNOWLEDGMENTS

This work was carried out under a grant from the Henry J. Kaiser Family Foundation, Menlo Park, CA (87-4361). Additional support was provided by the National Cancer Institute (90-2118-06). We wish to thank Dennis DuVall, Ginny Scobba, and Jane Steetle for their careful field work.

REFERENCES

1. Ernst ND, Wu M, Frommer P, Katz E, Matthews O, Moskowitz J, Pinsky JL, Pohl S, Schreiber GB, Sondik E, *et al*: Nutrition education at the point of purchase: the foods for health project evaluated. Prev Med 1986; 15(1):60–73.
2. Jacobs DR Jr, Luepker RV, Mittelmark MB, Folson AR, Pirie PL, Mascioli SR, Hannan PJ, Pechacek TF, Bracht NF, Carlaw RW, *et al*: Community-wide prevention strategies: evaluation design of the Minnesota Heart Health Program. J Chronic Dis 1986; 39(10):775–788.
3. Lasater TM, Lefebvre RC, Carleton RA: The Pawtucket Heart Health Program: Community level programming for heart health. RI Med J 1988; 71(1):31–34.
4. Kottke TE, Puska P, Salonen JT, Tuomilehto J, Nissinen A: Changes in perceived heart disease risk and health during a community-based heart disease prevention program: the North Karelia project. Am J Public Health 1984; 74:1404–1405.
5. Wallack LM: Mass media campaigns: the odds against finding behavior change. Health Educ Q 1981; 8:209–260.
6. Bartko JJ: The intraclass correlation coefficient as a measure of reliability. Psychol Rep 1966; 19:3–11.
7. Fleiss JL: Statistical Methods for Rates and Proportions, 2nd Ed. New York: John Wiley, 1981.

Poison Control Center Follow-up of Occupational Disease

2T

EDDY A. BRESNITZ, MD, MS

Abstract: We followed up 73 of 372 calls to a Regional Poison Control Center (RPCC) that involved workplace disease/exposure(s); most other calls were not made by the workers. An average of 12 additional people per workplace were potentially exposed. Six of the 73 contacted a government agency for investigation of the hazard/illness. Twenty-five percent of callers were still exposed an average of seven months after the original call. The results indicate that poison control centers should develop a public health component to calls about possible workplace poisonings. (*Am J Public Health* 1990; 80:711–712.)

Introduction

An accurate estimate of the incidence of occupational disease in the United States is hampered by the lack of an active surveillance system.[1–4] Investigators have proposed using existing data systems including regional poison control centers[5,6] for passive surveillance of occupational disease. A call may represent a group of affected/exposed workers and serve as a sentinel health event that identifies hazardous worksites.[7]

We conducted a follow-up survey of individuals with occupational exposure/disease who contacted the Delaware Valley Regional Poison Control Center (DVRPCC), over a 12-month period.

Methods

Approximately 7,760 individuals over the age of 17 contacted the Delaware Center between July 1, 1986 and June 30, 1987. Information was recorded on the American Association of Poison Control Centers' precoded report form. Referral guidelines listing the telephone numbers of outside agencies to which occupationally related callers may be referred are available to staff. The guidelines indicate that calls or complaints about known Occupational Safety and Health Administration (OSHA) regulated substances should be referred to OSHA and complaints about substances not covered or enforced by OSHA should be referred to the National Institute for Occupational Safety and Health (NIOSH).

A workplace exposure was recorded on the intake form of 372 calls (4.8 percent). Trained personnel, unrelated to the poison control center, abstracted data from these intake forms and called the telephone number obtained during the initial encounter. These follow-up calls were made between July 1 and November 1, 1987, an average of seven months after the initial call.

The interviewer explained the purpose of the study to the affected worker and obtained informed consent. To proceed, the interview was then either administered immediately or within one week at a prearranged time. In three cases, the interviewee was the sentinel case's employer who answered the questions from his/her perspective. Interviewers used a pre-tested, pre-coded form containing predominantly closed-ended questions. The analysis included 95% confidence intervals for differences in proportions.[8]

The study protocol was reviewed and approved by the Committee for the Protection of Human Subjects of the Medical College of Pennsylvania and the Medical Advisory Panel of the DVRPCC.

Results

The interviewers successfully interviewed 73 of the 372 work-related calls. Table 1 compares the characteristics of the 73 workers interviewed and the 299 workers not interviewed from information on the Center's intake form. Of those not interviewed, 209 (69.9 percent) calls originating from medical personnel or hospitals were excluded, because we did not believe that our questions would be answered without the patient's consent. Forty-nine callers (16.4 percent) either left no telephone number or gave a disconnected or false number. Of the remainder, 16 (5.4 percent) had been terminated from their jobs, 15 (5 percent) refused to answer questions and one (.3% percent) was an informational call.

The interviewed workers (responders) had acute exposures to more than 60 different chemicals at 73 different worksites; 19 individuals were exposed to two or more substances. All but four of the affected workers were symptomatic at the time of the initial call.

The responders had worked an average of 5.5 years at the worksite where the exposure occurred at the time of the follow-up call. Forty-nine (67 percent) of the 73 responders indicated that other workers (mean of 12) had been exposed

---

Address reprint requests to Eddy A. Bresnitz, MD, Division of Occupational and Environmental Health, Department of Community & Preventive Medicine, Medical College of Pennsylvania, 3300 Henry Avenue, Philadelphia, PA 19129. This paper, submitted to the Journal December 27, 1988, was revised and accepted for publication November 8, 1989.

TABLE 1—Comparison of Interviewed and Non-Interviewed Workers Based on Information from Poison Control Center (PCC) Intake Form

Characteristics	Exposed Workers Interviewed (N = 73)	Exposed Workers Not Interviewed (N)	95% CI of Difference
Mean Age (years)	30.8	30.5 (229)	
Sex (%)			
Male	66	78 (277)	(−23.9, 0.1)
Female	34	22	
Site of Caller (%)			
Residence	63	13	(38, 62;
Workplace	34	15 (289)	(7, 31)
Health Care Facility	3	72	(−75, −63)
Hospitalized (%)	0	6 (186)	(−12, −4)
PCC called back within 24 hours (%)			
Yes	69	59	(−2, 22)
No	31	37 (288)	
Tried and Failed	0	3	
Referral to Other Non-Medical Agency (%)			
Yes	6	15 (246)	(−16, −2)
No	94	85	
Route of Exposure			
Inhalation	67	42	(13, 37)
Ocular	11	32 (289)	(−30, −12)
Dermatologic	21	20	(−9, 11)
Ingestion	1	6	(−9, 11)
Parenteral	0	0	

at their worksite. Eight (16 percent) of these 49 responders indicated that one or more co-workers had been out on sick leave for the same exposure.

Overall, 48 responders reported they were advised to call their physician; 75 percent of these complied with this advice. Poison center staff recorded referrals to outside agencies in only four of the 73 calls (5 percent). However, 10 (14 percent) responders stated they were advised to call either OSHA, NIOSH, or the local health department. Five of seven reported that they called OSHA. One receiving a recommendation to call the health department or NIOSH did so.

Twenty-five of the 47 responders who approached their employer reported the institution of one or more primary preventive measures. Seven responders indicated that they were either fired or harassed or transferred.

Table 2 describes other issues assessed in the study. Only 37 percent of the responders had heard of either municipal, state, or federal right-to-know legislation, most frequently in the workplace or in the media. A responder was more likely to have heard about right-to-know laws if he/she had been hired less than one year prior to their initial call (62.5 vs 30 percent, 95% CI of difference = 5, 60).

TABLE 2—Other Public Health Issues Assessed in Responding Workers at Follow-Up

Health Issues (N)	Yes % (N)	95% CI
Worker still exposed (73)	25 (18)	16, 36
Existence of workplace union (70)	23 (16)	14, 34
Health and safety person present in workplace at time of exposure (e.g., employer, maneger) (69)	49 (35)	37, 61
Worker still having health problems (72)	4 (3)	1, 12
Suffered disability or work loss (72)	32 (23)	21, 41
Sick leave covered by Workers' Compensation (23)	22 (5)	8, 44
Currently seeing physician (71)	4 (3)	1, 12
Caller heard about right-to-know law (73)	37 (27)	26, 49
Caller read material safety data sheet (69)	17 (12)	9, 28

Discussion

The data reveal that poison center staff infrequently referred occupationally related calls to agencies concerned with occupational health despite existing guidelines and may not have routinely documented such information. Referred callers tended to follow poison center staff advice when given.

Blanc and Olson[5] determined that many more occupationally related cases were enumerated through a state-mandated physician-initiated active reporting system than through a passive system such as a poison control center. Our study supports this finding.

Although we studied a selected sample of workers, the survey most likely underestimates the potential magnitude of the problem. The majority of those not reached were workers evaluated in a health care facility or admitted to hospitals. A greater percentage of those not reached were hospitalized, indicating more severe medical problems and, perhaps, more serious workplace hazards in this group.

Poison control centers reported that 6.8 percent of approximately 312,000 calls from adults in 1987 were related to exposures in the workplace.[9] Although this data base appears to be a source of supplementary surveillance data for occupational illness, if our experience represents that of other centers, the staff may need to modify their protocols and improve their public health approach to callers with occupationally related illness/exposures. Centers could refer all workplace-related calls to a separate state or federal agency responsible for hazard evaluation and follow-up. Failure to investigate these calls may result in continued exposure and illness in all workers connected to an index case.

ACKNOWLEDGMENTS

The author expresses gratitude to Sally Kirkpatrick and Kate Kraft for their assistance in data collection and coding; to Ed Gracely for statistical analysis and critical review; and to Carolyn Needleman, Kathleen M. Rest, Harriet Rubenstein, Thomas Kearney, and Fred Henretig for their helpful comments. This study was presented at the 116th American Public Health Association Annual Meeting, Boston, MA on November 16, 1988.

REFERENCES

1. National Academy of Sciences/National Research Council, Committee on National Statistics: Counting Injuries and Illnesses in the Workplace: Proposals for a Better System. Washington, DC: National Academy Press, 1987.
2. US Congress, Office of Technology Assessment: Preventing Illness and Injury in the Workplace. Pub. No. OTA-H-256. Washington, DC: OTA, April 1985.
3. Committee on Government Operations, US House of Representatives: Occupational illness data collection: fragmented, unreliable, and seventy years behind communicable disease surveillance. Report No. 98-1144. Washington, DC: Supt of Documents, October 5, 1984.
4. Wegman DH, Froines JR. Surveillance needs for occupational health. (editorial) Am J Public Health 1985; 75:1259–1261.
5. Blanc PD, Olson KR: Occupationally related illness reported to a regional poison control center. Am J Public Health 1986; 76:1303–1307.
6. Pepper LD, Brooks DR, Goldman R, *et al*: A model system for occupational disease intervention/prevention based on a regional poison control center (abstract). American Public Health Association, Official Program, 116th Annual Meeting, Boston, MA 1988.
7. Rutstein DD, Mullan RJ, Frazier TM, *et al*: Sentinel health events (occupational): A basis for physician recognition and public health surveillance. Am J Public Health 1983; 73:1054–1062.
8. Fleiss JL: Statistical Methods for Rates and Propositions. New York: John Wiley & Sons, 1981.
9. Litovitz T, Schmitz BF, Matyunas N, *et al*: 1987 Annual report of the American Association of Poison Control Centers National Data Collection System. Am J Emerg Med 1988;6:479–520.

11. Goitein RK, Sykes RL, Baer DM, Belsey RE: An evaluation of a physician office analyzer, The Boehringer Mannheim Reflotron. (Abstract) Clin Chem 1987; 33:936.
12. Hawks RL, Chiang CN, National Institute on Drug Abuse Research: Urine Testing for Drugs of Abuse. DHHS Pub. No. ADM 87-1481, Monograph #73. Washington, DC: Govt Printing Office, 1986.
13. Mandatory guidelines for federal workplace drug testing programs; Final Guidelines; Notice. Federal Register, April 11, 1988; 53:11970–11989.

Illegal Abortion in Mexico: Client Perceptions

17

SUSAN PICK DE WEISS, PHD, AND HENRY P. DAVID, PHD

Abstract: An exploratory study of the perceptions of 156 abortion clients in Mexico suggests that perceived quality of service was the main reason for choosing physicians while cost and anonymity were the major reasons for choosing nonphysicians. "Too young" was the most often cited reason for pregnancy termination, followed by economic situation and having too many children already. Cost was, on average, equivalent to three to four weeks minimum wage; physicians' charges were about three times higher than those of nonphysicians. (*Am J Public Health* 1990; 80:715–716.)

Introduction

In Latin America, abortion is legally restricted everywhere except in Cuba.[1] In Mexico, abortion is legally permitted only for reasons of health, rape, or incest.[2] Contraceptive services are widely available, especially in the cities.

The public health, economic, social, and psychological costs associated with the illegal termination of unwanted pregnancies have been repeatedly documented[3–6]. Complications of clandestine abortions presently constitute the fifth largest cause of maternal mortality and the third highest reason for admission to gynecological services in the metropolitan hospitals of the Mexican Social Security System; no reliable data are available on women admitted to other services.[7]

To obtain information on the perceptions of women seeking clandestine abortions in Mexico, an exploratory study was conducted in Mexico City, Acapulco, and Oaxaca. Another objective was to test the feasibility of conducting behavioral research in an area of public health that throughout much of the region is culturally taboo and severely restricted by law.

Methods

Twelve experienced female graduate and undergraduate psychology students, sensitive in conducting individual interviews, some of whom had had abortions, were trained by the senior author (SPdW) to seek out personal acquaintances who had terminated unwanted pregnancies illegally in the recent past. On completion of interviews the subjects were invited to suggest other possible study participants. Of 159 women known to have had at least one induced abortion in the recent past, 156 agreed to individual interviews. The questionnaire was composed of mostly open-ended items that had been field-tested. Interviewees were assured of anonymity and confidentiality of replies.

Results

Of the 156 women, 119 had their abortion performed by physicians, 32 by untrained service providers, and five could not recall their providers' qualifications. As shown in Table 1, of the women attended by a physician most were not married. Ages ranged from 12 to 56 years; clients attended by physicians were significantly older and better educated than those seen by others. More professional women were seen by physicians. Over one-third of the women had at least one child.

A lower proportion of clients attended by physicians had experienced two or more abortions compared to those seen by nonmedically trained persons. Most of the procedures performed by physicians occurred in a private clinic or hospital (91.6 percent), while other service providers generally operated in the client's home. Quality of service was the most frequent reason for going to physicians (30.8 percent), followed by security, while cost (56.2 percent) was the main reason for seeking other providers, followed by security and anonymity. As also shown in Table 1, when asked in an open-ended question without probe, the most often cited reason for having an abortion among all women was becoming pregnant at too young an age. Another major reason among physician-attended women was the partner's rejection of the pregnancy. Among women attended by untrained providers, the second and third reasons were economic and having too many children already.

No differences between the two groups were noted among cited referral sources which included friends, sexual partners, family members, physicians, and psychologists. About half the physician-attended women considered the location of the service far away from their home compared to 25 percent of those attended by untrained providers. The cost of a physician-induced procedure was, on average, the equivalent of three to four weeks legal minimum wage in Mexico. A physician-provided service was generally two to three times more expensive than that provided by others ($88 vs $33 in 1987/88). Among women attended by physicians 43 percent reported receiving contraceptive counseling in connection with their abortion, compared to only 3 percent of those who went to other providers.

As further shown in Table 1, for the women attended by physicians, the most often reported procedures were curettage; over one-fifth did not know what procedure had been

---

Address reprint requests to Henry P. David, PhD, Director, Transnational Family Research Institute, 8307 Whitman Drive, Bethesda, MD 20817. Findings in tabular form may be obtained from Susan Pick de Weiss, PhD, Instituto Mexicano de Investigacion de Familia y Poblacion, Apartado Postal 41–595, Mexico, D.F. 11001, Mexico. Dr. De Weiss is also on the Facultad de Psicologia, Universidad Nacional Autonoma de Mexico. This paper, submitted to the Journal January 3, 1989, was revised and accepted for publication September 1, 1989.

TABLE 1—Characteristics of Two Groups of Woman Who Had Induced Abortions, Mexico

Characteristics	Physician Attended (n = 119)	Other Attended (n = 32)	Difference	(95% CI)
Mean Age	26.2	24.5	1.7	(−.85, 4.25)
Mean No. of Children	1.97	2.06	−0.09	(−.9, .72)
Average Cost in Dollars	88	33	55	(45.2, 64.8)
	Percentages			
Single	67	59	8	(−9,25)
Attended College	39	25	14	(−9,37)
Professional Occupation	19	4	15	(8,22)
Two or More Abortions	13	22	−9	(−36, 18)
Received Contraceptive Information	43	3	40	(15, 65)
Post-abortion Problems	22	47	−25	(−53, 3)
	Procedure Used			
Curettage	50	13	37	(15, 59)
Catheters	3	19	−16	(−46, 14)
Injections	3	25	−22	(−51, 7)
Suction	20	–	–	(–)
Wire, Needles, Herbs	–	29	–	(–)
Unknown to Woman	24	13	11	(−17, 39)
	Reason for Abortion			
Too Young	31	34	−3	(−27, 21)
Partner Pressure	39	13	26	(1, 51)
Too Many Children	8	16	−8	(−38, 22)
Economic	15	19	−4	(−32, 24)
Other Reasons	7	18	−11	(−41, 19)

used. Among the women attended by untrained service providers, the most often cited procedures were injections, catheters, herbs, curettage, and wires or needles; few did not know what procedure was used.

Before going to a service provider, almost two-thirds of all the women had purchased pseudoabortifacients without adequate knowledge of possible risks. About one-third of all the women had tried inducing an abortion themselves, resorting to herbs, injections, or a combination of both, or of either with pills, hard exercise, or the introduction of objects into the cervix. Post-abortion complications were noted by less than one-fifth of the physician-attended women (mostly cramps) compared to nearly half of those attended by untrained providers who noted more hemorrhages (22 percent vs 47 percent). When asked if they would recommend their provider to other women, three-fourth of the physician-attended women replied affirmatively compared to one-fourth of those attended by other service providers.

Discussion

It is recognized that the findings from this exploratory study are limited by the nonrepresentativeness of the sample. However, the responses obtained confirm informal impressions that nonmedically trained persons provide abortion services primarily to women of lower socioeconomic and educational levels, and that such women are at greater risk for experiencing post-abortion complications and for future unwanted pregnancies since contraceptive counseling is seldom provided.

The method of open-ended interviewing developed for this exploratory study appears suitable for further adaptation for behavioral research on the sensitive topic of abortion with more representative populations in those countries of the region where the procedure remains a taboo topic. In concert with more epidemiological research, such studies may be expected to contribute to a reexamination of the public health risks of legally restricted abortion and to motivate improved access to effective contraception that will reduce the future incidence of unwanted pregnancies.

ACKNOWLEDGMENTS

The authors wish to thank the UNAM (Universidad Nacional Autonoma de Mexico) psychology students who participated in data collection, Patricia Andrade-Palos for her comments on the manuscript, and the Population Crisis Committee for partial support of the study.

REFERENCES

1. David HP, Pick de Weiss S: Abortion in the Americas. *In*: Pan American Health Organization: Reproductive Health. Washington, DC: PAHO (in press).
2. Mexico. Penal Code of 2 January, 1931, Arts. 329–334.
3. Viel B: The health consequences of illegal abortion in Latin America. *In:* Landy U, Ratnam SS (eds): Prevention and Treatment of Contraceptive Failure. New York: Plenum, 1986.
4. David HP: Abortion policies. *In:* Hodgson JE (ed): Abortion and Sterilization: Medical and Social aspects. New York: Academic Press, 1981.
5. David HP. Abortion: its prevalence, correlates, and costs. *In:* Bulatao RA, Lee RD (eds): Determinants of Fertility in Developing Countries. New York: Academic Press, 1983.
6. Figa-Talamanca I: Abortion and mental health. *In:* Hodgson J (ed): Abortion and sterilization: medical and social aspects. New York: Academic Press, 1981.
7. Lovera S: No es una cuestion moral, sino de salud publica y social. *La Jornada* (Mexico City), March 31, 1989.

2T

A Survey of State Medicaid Policies for Coverage of Abortion and Prenatal Diagnostic Procedures

JANET WEINER, MPH, AND BARBARA A. BERNHARDT, MS

Abstract: In the summer of 1988, we surveyed all states to evaluate access to Medicaid funding for abortion after the diagnosis of an anomalous fetus. All state Medicaid programs covered amniocentesis, and most had expanded coverage to include newer prenatal diagnostic procedures for eligible women. In 29 states, however, abortion coverage was limited to instances when continuation of abortion threatens the life of the mother. Only 13 states paid for a woman on Medicaid to obtain an abortion after diagnosis of an anomalous fetus. (*Am J Public Health* 1990; 80:717–720.)

Introduction

Medicaid is a joint state- and federally-funded program, enacted in 1965, to provide health care coverage for many indigent and disabled citizens. Within broad federal guidelines, each state has latitude in deciding which medical and ancillary services are to be covered by its Medicaid program.

In 1973, the US Supreme Court decision in *Roe v. Wade* prohibited states from interfering with a woman's right to abortion except to protect maternal health or a viable (third trimester) fetus. By 1974, three-fourths of the states covered most abortions under their Medicaid programs.[1]

In 1977, Congress passed the Hyde Amendment, which limited federal reimbursement of abortions to situations that threatened the life or physical health of the mother, and to victims of rape and/or incest. Current federal policy reimburses states only for abortions performed to save the life of the mother. Abortions for other reasons, which constitute over 99 percent of all abortions, must be paid for by non-federal or, in most cases, state funding.[2]

At present, increasingly sophisticated techniques such as amniocentesis, chorionic villus sampling (CVS), ultrasound, and biochemical assays are permitting earlier and broader detection of fetal abnormalities. Malpractice fears impel physicians to identify patients at risk for producing children with prenatally-diagnosable defects and offer them appropriate testing.[3] At least one state requires that maternal serum alpha-fetoprotein (MSAFP) screening be offered to pregnant women.[4] Ethical practice dictates that when tests show a fetal anomaly exists, the woman be offered the option of pregnancy termination, within the guidelines of each state's law regulating abortion.[5] Information has been unavailable nationally on coverage of prenatal diagnostic services by Medicaid programs and on utilization of these procedures by pregnant Medicaid recipients.

The survey reported here delineates state-by-state Medicaid guidelines for coverage of prenatal diagnostic procedures and abortion.

Methods

We developed a questionnaire that asked about Medicaid coverage of seven prenatal services: amniocentesis, ultrasound to rule out fetal anomalies, CVS, MSAFP testing, fetal DNA analysis, chromosome analysis on cultured amniotic fluid cells, and genetic counseling. We included the standard Current Procedural Terminology Codes for each procedure (when available). Respondents were asked to check either "covered", "not covered", "covered on a case-by-case-basis", or "unknown" for each procedure. We asked about Medicaid coverage of abortion for seven indications, broad enough to encompass the variation in state policies, using the legal terminology of indications for abortion as reported in the Medicare and Medicaid guide.[6]

We mailed the questionnaire to the director of the Medicaid program in each state and the District of Columbia. After follow-up, including telephone calls, the final response rate was 100 percent. We did not attempt to verify how completely these guidelines were followed in each state. The responses reflect Medicaid regulations as of summer 1988.

Results

Each state's coverage of prenatal diagnostic procedures is shown in Table 1. The majority of states cover all procedures listed, and almost all have developed guidelines for coverage, as indicated by the low numbers of "case-by-case" or "unknown" responses. All states and the District of Columbia cover amniocentesis, and all but one cover MSAFP testing. Two-thirds cover CVS and three-fourths cover DNA analysis, both of which are newer technologies for the detection of fetal abnormalities.

Table 2 lists each state's coverage for abortion. At the time of the survey, seven states and the District of Columbia paid for abortion on demand. All states cover abortions that save the life of the mother, the only ones for which federal reimbursement is available. In 29 states, this is the *only* indication for which Medicaid will cover an abortion. Eleven states will pay for abortion when the indication is a known or suspected fetal anomaly, and two other states evaluate coverage on a case-by-case basis. Just over one-fourth of the states will pay for "medically necessary" abortions, usually as certified by an attending physician. Likewise, victims of rape and/or incest are covered for abortion in one-fourth of the states. This indication has been hotly debated on a federal level, as President George Bush recently vetoed a congressional package that would have reinstated federal reimbursement for abortion in cases of rape and incest.

Discussion

A combination of Medicaid regulations has led to the incongruous policy in many states of covering prenatal diagnostic services but not the selective abortion in cases of a diagnosed fetal abnormality. We believe that these policies

---

From the Division of Medical Genetics, Johns Hopkins School of Medicine (Weiner) and the Department of Women's Services and Pediatrics, Sinai Hospital, Baltimore, MD (Bernhardt). Address reprint requests to Janet Weiner, MPH, Department of Scientific Policy, American College of Physicians, 6th Street at Race, Philadelphia, PA 19106-1572. This paper, submitted to the Journal June 29, 1989, was revised and accepted for publication October 12, 1989.

TABLE 1—Medicaid Coverage of Prenatal Diagnostic Procedures by States and District of Columbia as of September 1988

States	Amniocentesis	Fetal Ultrasound	CVS	MSAFP	DNA Analysis	Karyotyping	Genetic Counseling
Alabama	X	X	X	X	X	X	X
Alaska	X	X	X	X	X	X	X
Arizona	X	X	X	X	C	X	X
Arkansas	X	X	C	X	X	X	X
California	X	X	0	X	0	X	X
Colorado	X	X	0	X	?	X	X
Connecticut	X	X	?	X	X	0	?
Delaware	X	X	X	X	X	X	X
District of Columbia	X	X	C	X	0	X	X
Florida	X	0	X	X	X	X	?
Georgia	X	X	X	X	X	X	?
Hawaii	X	0	C	X	..	X	X
Idaho	X	X	X	X	X	X	0
Illinois	X	X	X	X	X	X	X
Indiana	X	X	X	X	X	X	X
Iowa	X	0	X	X	X	C	C
Kansas	X	0	0	X	X	0	0
Kentucky	X	X	X	X	X	X	X
Louisiana	X	X	0	X	X	X	X
Maine	X	?	X	X	X	X	X
Maryland	X	X	X	X	C	X	X
Massachusetts	X	X	C	X	X	X	X
Michigan	X	X	X	X	X	X	X
Minnesota	X	X	X	X	?	X	X
Missouri	X	0	0	X	0	X	X
Mississippi	X	X	C	X	C	X	X
Montana	X	X	X	X	X	X	X
Nebraska	X	X	X	X	X	X	X
Nevada	X	X	X	X	X	X	X
New Hampshire	X	X	X	X	X	X	X
New Jersey	X	X	C	X	C	X	X
New Mexico	X	X	X	X	X	C	X
New York	X	X	X	X	0	X	X
North Carolina	X	X	0	X	X	0	0
North Dakota	X	X	X	X	C	X	X
Ohio	X	X	X	X	X	X	X
Oklahoma	X	C	0	0	0	0	0
Oregon	X	X	X	X	X	X	0
Pennsylvania	X	0	0	X	X	X	X
Rhode Island	X	X	C	X	C	C	X
South Carolina	X	X	X	X	X	X	X
South Dakota	X	X	X	X	X	X	X
Tennessee	X	X	X	X	X	X	X
Texas	X	X	0	X	X	X	X
Utah	X	X	X	X	X	X	X
Vermont	X	X	X	X	X	X	X
Virginia	X	X	X	X	X	X	X
Washington	X	X	X	X	X	X	0
West Virginia	X	X	X	X	X	X	X
Wisconsin	X	X	X	X	X	X	X
Wyoming	X	X	X	X	X	X	X
Totals							
Covered	51	43	34	50	38	44	42
Not Covered	0	6	9	1	5	4	6
Case-by-case	0	1	7	0	6	3	1
Unknown	0	1	1	0	2	0	3

Symbols
X = covered
0 = not covered
C = case-by-case
? = unknown

discriminate against poor women and interfere with ethical medical practice. By covering prenatal diagnostic services in Medicaid programs, each state in this country acknowledges the medical necessity of these services and its commitment to providing access to them. Since abortion is a recognized option for women carrying an anomalous fetus, states that refuse to pay for abortion interfere with women's rights. Once a state has agreed to pay for diagnostic procedures, we do not believe it should influence subsequent medical decisions.

In the future, Medicaid guidelines will affect more and more women, as Congress encourages states to expand eligibility criteria for pregnant women. By July 1990, federal Medicaid guidelines mandate that all states cover pregnant women at poverty level, and will give states the option of covering women with incomes up to 185 percent of poverty

TABLE 2—Medicaid Coverage of Abortion Services by States and the District of Columbia as of September 1988

State	On Demand	Life-threatening	Physical Health Damage	Mental Health Damage	Fetal Anomaly	Medical Necessity	Rape/Incest
Alabama	0	X	0	0	0	0	0
Alaska	X	X	X	X	X	X	X
Arizona	0	X	0	0	0	0	0
Arkansas	0	X	0	0	0	0	0
California	X	X	X	X	X	X	X
Colorado*	0	X	C	C	C	C	0
Connecticut	0	X	C	C	C	X	C
Delaware	0	X	0	0	0	0	0
District of Columbia*	X	X	X	X	X	X	X
Florida	0	X	0	0	0	0	0
Georgia	0	X	0	0	0	0	0
Hawaii	X	X	X	X	X	X	X
Idaho	0	X	C	C	0	C	0
Illinois	0	X	0	0	0	0	0
Indiana	0	X	0	0	0	0	0
Iowa	0	X	0	0	X	0	X
Kansas	0	X	0	0	0	0	0
Kentucky	0	X	0	0	0	0	0
Louisiana	0	X	0	0	0	0	0
Maine	0	X	C	C	0	C	0
Maryland	0	X	X	X	X	0	X
Massachusetts	X	X	X	X	X	X	X
Michigan*	X	X	X	X	X	X	X
Minnesota	0	X	0	0	0	X	X
Missouri	0	X	0	0	0	0	0
Mississippi	0	X	0	0	0	0	0
Montana	0	X	0	0	0	0	0
Nebraska	0	X	0	0	0	0	0
Nevada	0	X	0	0	0	0	0
New Hampshire	0	X	0	0	0	0	0
New Jersey	X	X	X	X	X	X	X
New Mexico	0	X	0	0	0	0	0
New York	0	X	X	X	0	X	0
North Carolina	0	X	0	C	0	0	0
North Dakota	0	X	0	0	0	0	0
Ohio	0	X	0	0	0	0	0
Oklahoma	0	X	0	0	0	0	0
Oregon	0	X	X	X	0	X	0
Pennsylvania	0	X	0	0	0	0	X
Rhode Island	0	X	0	0	0	0	0
South Carolina	0	X	0	0	0	0	0
South Dakota	0	X	0	0	0	0	0
Tennessee	0	X	0	0	0	0	0
Texas	0	X	0	0	0	0	0
Utah	0	X	0	0	0	0	0
Vermont	0	X	0	0	0	0	0
Virginia	0	X	X	X	0	X	0
Washington	X	X	X	X	X	X	X
West Virginia	0	X	X	X	X	X	X
Wisconsin	0	X	X	0	0	0	X
Wyoming	0	X	0	0	0	0	0
Totals							
Covered	8	51	14	13	11	14	14
Not Covered	43	0	33	33	38	34	36
Case-by-case	0	0	4	5	2	3	1

Symbols
X = covered
0 = not covered
C = case-by-case

*Since the fall of 1988, Michigan and Colorado, by voter referenda, and the District of Columbia, by legislative action, altered their regulations to pay only for life-threatening abortions.

level.[7] Therefore, the public health implications of Medicaid policies on abortion will become even greater.

As budgetary pressures force state and federal agencies to cut costs, the economic burden of restrictive abortion funding will loom larger. The financial cost of the Hyde Amendment was estimated conservatively to be $95 million in 1986. For every dollar spent on a desired abortion through Medicaid, four dollars are saved in medical and social welfare costs in just two years.[8] The medical and emotional costs of bearing children that are either not wanted or have major anomalies may be far greater.

Health care costs of infants and children with genetic disorders and chronic illnesses are at least three times higher than those of other children.[9] Those born to low-income families pose an even greater challenge to an already overburdened household. Children with disabilities require additional health care services including occupational and physical therapy, psychosocial support, home health care, respite and day care.

On a state level, Medicaid policies on abortion have been heavily influenced by political pressure from right-to-life groups. Since our survey, two states (Colorado and Michigan) have altered their regulations because of referenda placed on the ballot in the November 1988 elections, and the District of Columbia has withdrawn funding entirely. Regardless of one's views on abortion, we believe the Medicaid program should not be used to force the poorest women among us to bear children with severe congenital anomalies.

ACKNOWLEDGMENTS

This study was presented at the annual meeting of the American Society of Human Genetics in October 1988.

REFERENCES

1. Torres A, Donovan P, Dittes N, Forrest J: Public benefits and cost of government funding for abortion. Fam Plann Perspect 1986; 18:111–118.
2. Gold RB, Guardado S: Public funding of family planning, sterilization and abortion services, 1987. Fam Plann Perspect 1988; 20:228–233.
3. Rhodes AM: Legal aspects of prenatal diagnosis. Clin Obstet Gynecol 1988; 31:233–252.
4. Steinbrook R: In California, voluntary mass prenatal screening. Hastings Cent Rep 1986; 16:5–7.
5. Powledge TM, Fletcher J: Guidelines for the ethical, social and legal issues in prenatal diagnosis. N Engl J Med 1979; 300:168–172.
6. Medicare and Medicaid Guide, Vol. 3. Chicago: Commerce Clearing House, Inc. ¶ 15,500–15,660.
7. P.L. 100-360, Sec. 302.
8. Kenney A, Torres A, Dittes N, Macias J: Medicaid expenditures for maternity and newborn care in America. Fam Plann Perspect 1986; 18:103–109.
9. Newacheck PA, McManus MA: Financing health care for disabled children. Pediatrics 1988; 81:385–394.

Obstetrical Pulmonary Embolism Mortality, United States, 1970–85

ADELE L. FRANKS, MD, HANI K. ATRASH, MD, MPH, HERSCHEL W. LAWSON, MD, AND KAREN S. COLBERG, BA

Abstract: To investigate pulmonary embolism as a cause of obstetrical death, vital records data from 1970 through 1985 were analyzed. Results showed that the number of obstetrical pulmonary embolism deaths per 100,000 live births declined by 50 percent for both Whites and Blacks. However, Black women maintained more than a 2.5-fold higher risk, and women over age 40 had a ten-fold higher risk of embolism mortality. Thus, although the risk of obstetrical pulmonary embolism death has declined, some subgroups of women remain at higher risk. (*Am J Public Health* 1990; 80:720–721.)

Introduction

Several studies of maternal mortality have found pulmonary embolism to be the leading cause of maternal death in recent years.[1–5] It has been suggested that pulmonary embolism deaths may be becoming more important as a cause of maternal death because deaths due to other causes are declining more rapidly, or because medicolegal concerns favor the diagnosis of causes of death perceived to be nonpreventable.[1,4] Little is known nationally about trends in maternal mortality due to pulmonary embolism.

The purpose of the current analysis was to determine the absolute and relative importance of pulmonary embolism as a cause of obstetrical death over time on a national basis, and to describe the demographic risk factors for obstetrical pulmonary embolism (OPE) death.

Methods

The total number and characteristics of obstetrical deaths were obtained from multiple cause of death mortality tapes made available by the National Center for Health Statistics (NCHS). Up to 20 diagnoses are recorded from each death certificate and coded according to the International Classification of Diseases (ICD).[6,7] Obstetrical deaths were identified as those with any nonabortive pregnancy-related diagnosis (ectopic pregnancies, spontaneous abortions, induced abortions, and molar pregnancies were excluded). For the years 1970 through 1978 OPE deaths included ICDA-8 codes 634 and 673, while for 1979–85 they included ICD-9-CM code 673. These data do not allow one to separately identify amniotic fluid, thrombotic or air pulmonary embolism; therefore all causes of pulmonary embolism are grouped together.

The OPE mortality ratio is defined as the number of obstetrical deaths associated with pulmonary embolism per 100,000 live births during the same time period. The numbers of live births for each year were obtained from national natality computer tapes made available by NCHS. The OPE mortality ratios for both Blacks and Whites over time were standardized to the age distribution of White live births 1970–85.

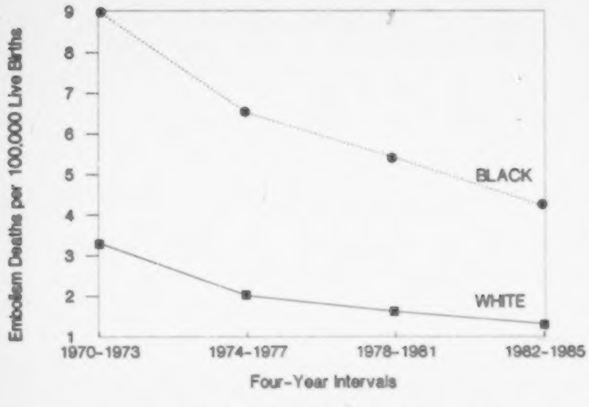
The percent of obstetrical deaths due to embolism is the number of pulmonary embolism-associated obstetrical deaths per 100 obstetrical deaths during the same time period. The analysis is restricted to women over the age of 14 years and younger than 45 years who were classified as either White or Black.

Results

From 1970 through 1985 there were a total of 1,417 OPE deaths and 55,096,188 live births, for an overall OPE mortality ratio of 2.6 OPE deaths per 100,000 live births. Over the 16-year interval OPE mortality ratios, standardized by age, declined more than 50 percent for both Whites (from 3.3 to 1.3 per 100,000 live births) and for Blacks (from 9.0 to 4.2 per 100,000 live births) (Figure 1). However, Black women maintained more than a 2.5-fold higher risk of OPE mortality than White women throughout the 16-year interval. This racial disparity did not vary systematically over time or

---

Address reprint requests to Adele L. Franks, MD, Division of Reproductive Health, Pregnancy and Infant Health Branch, Center for Chronic Disease Prevention and Health Promotion, Centers for Disease Control, Mailstop C06, Atlanta, GA 30333. Drs. Atrash and Lawson are also with that Branch at CDC; Ms. Colberg is with Statistics and Computer Resources Branch, DRH, CCDPHP, CDC, Atlanta. This paper, submitted to the Journal September 22, 1989, was revised and accepted for publication December 4, 1989.



*Standardized to age distribution of live births to White women, 1970–1985

FIGURE 1—Obstetrical Pulmonary Embolism Deaths per 100,000 Live Births,* United States, 1970–85

between age groups. The percentage of all obstetrical deaths which were associated with pulmonary embolism did not differ between White and Black women, and remained relatively constant over time at approximately 25 percent.

Aggregating all 16 years under analysis, the absolute risk of OPE death increased with age for both race groups and was at least 2.5 times higher for Black women than for White women at all ages (Figure 2). For both racial groups, the risk for women 40 years of age or older was nearly 10 times greater than for women less than age 25.

Discussion

Although recent studies have found pulmonary embolism to be the leading cause of maternal death, vital records data show that the absolute risk of OPE death has been dropping and the proportion of obstetrical deaths associated with pulmonary embolism has not been increasing. Thus the belief that maternal pulmonary embolism deaths are a growing problem in the US is not supported by vital records data.

The limitations of vital records data in the surveillance of maternal death have been previously described.[8] Investigations designed specifically to identify and evaluate maternal deaths uncover more deaths, and often have more available information for assigning a cause of death. Indeed, vital records data do not allow clinical differentiation of immediate cause of death from underlying conditions which may contribute to death. We reviewed concurrent diagnoses from the death certificates of the OPE deaths under analysis. The most common concurrent diagnoses were cardiovascular disease, arrhythmia, shock or cardiac arrest (40 percent), hemorrhage or coagulopathy (17 percent), venous disease (9 percent), or "other ill-defined causes of morbidity or mortality" (9 percent). As these conditions could either precede or follow an OPE, inferences about which condition was the principal cause of death versus which conditions may have contributed to causing death cannot be made based on these data. However, we found no change over time in the distribution of concurrent diagnoses. In addition, we repeated our analysis using the single underlying cause of death as determined by NCHS. Results revealed trends over time and by race and age which were identical to those reported in this paper.

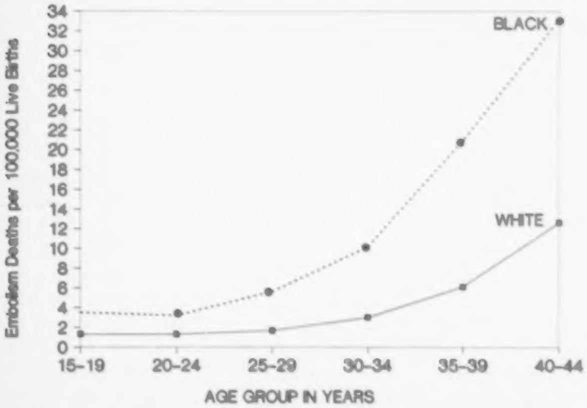


FIGURE 2—Obstetrical Pulmonary Embolism Deaths by Age and Race, United States, 1970–85

In this study we were unable to evaluate type of pulmonary embolism, although previous studies of maternal death have found half or more of maternal pulmonary embolism deaths to be thrombotic in nature.[1,3–5,9] Among causes of embolism, thrombotic events may be the most amenable to prevention through changes in medical/surgical practices.[10,11] Further research is necessary to characterize the temporal relationship between delivery and thrombotic events, as well as to identify potential risk factors for thrombotic death, such as method of delivery (vaginal vs cesarean section), personal characteristics (e.g., obesity), or underlying medical, surgical, or obstetrical conditions. With this information, interventions such as prophylactic use of pressure gradient stockings or anticoagulation[3] may be implemented and evaluated for those at highest risk. Unfortunately, vital records data alone cannot provide the increased clinical detail necessary to guide risk reduction strategies for this leading cause of obstetrical death in the United States; therefore much depends on current efforts to gather supplemental information about maternal deaths.[12]

REFERENCES

1. Kaunitz AM, Hughes JM, Grimes DA, Smith JC, Rochat RW, Kafrissen ME: Causes of maternal mortality in the United States. Obstet Gynecol 1985; 65:605–611.
2. Rochat RW, Koonin LM, Atrash HK, Jewett JJ: Maternal mortality in the United States: Report from the maternal mortality collaborative. Obstet Gynecol 1988; 72:91–97.
3. Sachs BP, Brown DAJ, Driscoll SG, Schulman E, Acker D, Ransil BJ, Jewett JJ: Maternal Mortality in Massachusetts: Trends and prevention. N Engl J Med 1987; 316:667–672.
4. Gabel HD: Maternal mortality in South Carolina from 1970 to 1984: An analysis. Obstet Gynecol 1987; 69:307–311.
5. McLean R, Mattison ET, Cochrane NE: Maternal mortality study annual report 1970–1976. NY State J Med 1979; 79:39–46.
6. National Center for Health Statistics: International classification of diseases, adapted for use in the United States. Eighth revision. PHS pub. no. 1693. Washington, DC: US Department of Health, Education, and Welfare, Public Health Service, 1968.
7. Commission on Professional and Hospital Activities: The international classification of diseases, 9th revision, clinical modification. DHHS pub. no. (PHS) 80-1260. Ann Arbor, MI: US Department of Health and Human Services, Public Health Service, Health Care Financing Administration, 1981.
8. Smith JC, Hughes JM, Pekow PS, Rochat RW: An assessment of the incidence of maternal mortality in the United States. Am J Public Health 1984; 74:780–783.
9. Moses V, DePersio SR, Lorenz D, Oberle MW, Rochat R, Fermo A: A thirty-year review of maternal mortality in Oklahoma, 1950 through 1979. Am J Obstet Gynecol 1987; 157:1189–1194.

10. Kakker VV, Corrigan TP, Fossard DP: Prevention of fatal postoperative pulmonary embolism by low doses of heparin. Lancet 1975; 2:45–51.
11. Gallus AS, Hirsh J, Tuttle RJ, Trebilcock R, O'Brien SE, Carroll JJ, Minden JH, Hudecki SM: Small subcutaneous doses of heparin in prevention of venous thrombosis. N Engl J Med 1973; 288:545–551.
12. Ellerbrock TV, Atrash HK, Hogue CJR, Smith JC: Pregnancy mortality surveillance: A new initiative. Contemp Obstet Gynecol June 1988; 31:23–34.

Cervix and Breast Cancer Incidence in Immigrant Caribbean Women

RACHEL G. FRUCHTER, PHD, MPH, KAMRAN NAYERI, MA, JEAN CLAUDE REMY, MD, CAROLYN WRIGHT, MS,
JOSEPH G. FELDMAN, DPH, JOHN G. BOYCE, MD, AND WILLIAM S. BURNETT, MD

Abstract: Cervix and breast cancer incidence in 1978–82 was computed for immigrant and United States-born Black women in Brooklyn, New York. Compared to the national SEER (Surveillance, Epidemiology and End Results) rates, US-born and Haitian women had high rates of invasive cervical cancer, while English-speaking Caribbean immigrants had an average rate. However, while US-born women had an average rate of carcinoma in situ of the cervix, both immigrant groups had low rates. Both immigrant groups had low rates of breast cancer, whereas US-born Black women had an average rate. (*Am J Public Health* 1990; 80:722–724.)

Introduction

Immigrant populations challenge the United States systems for preventive health care because they may differ from the US-born in their patterns of disease and in their knowledge and utilization of preventive health services. Where health statistics are recorded only by race, differences within populations may be obscured and information inadequate to plan effective programs.

In Brooklyn, New York City, about 20 percent of the Black community of about 700,000 are immigrants, primarily from the Caribbean basin. Haitian immigrants with invasive cancer of the cervix are diagnosed at a significantly later stage than US-born Black women,[1] and Haitians are less likely than US-born or English-speaking Caribbean women to have had a Pap test or to practice breast self-examination.[2] We determined the incidence of breast and cervix cancer among ethnic subgroups of the Black community in Brooklyn in order to help plan effective cancer prevention activities.

Methods

Population data from the 1980 US census were obtained from a 5 percent Public Usage Microdata Sample for Brooklyn. Birthplace was categorized as: 1) USA; 2) English-speaking Caribbean islands; 3) Haiti; and 4) Other. "Other" birthplaces included Africa, Europe, Canada, central and south America, and Spanish-speaking Caribbean islands. Birthplace was available for 92 percent of Black foreign-born women. For each five-year age group, foreign-born women of unspecified birthplace were allocated to above categories 2–4 in proportion to women of known birthplace. Cancer cases in Brooklyn residents from 1978–82 were obtained from the New York State Cancer Registry.[3] Cases were histologically confirmed in 93–99 percent depending on diagnosis.[3] Birthplace was not available for 8–14 percent of cases and was allocated as described for population data.

Although carcinoma in situ may be diagnosed in ambulatory facilities and therefore has potential for underregistration, low-income populations are usually dependent on hospital-based facilities and there is no reason to assume differential registration for immigrant and non-immigrant women.

For each group of Brooklyn women, we computed the age-standardized incidence ratio (SIR—observed:expected cases) comparing each group to the total Black US rates. We used the "indirect method" by applying the five-year age-specific rates for Black women from the 1978–81 data of the Surveillance, Epidemiology and End Results (SEER) program to the Brooklyn populations to obtain the expected numbers of cases.[4] The 95% confidence intervals (CI) of the SIR were computed from a Poisson distribution.[5] The indirect method was used because Haitian and "Other" groups had less than 50 cancer cases per site giving unstable age-specific rates.

The 1980 census undercounted low-income minority populations especially in areas with many immigrants.[6] Incidence ratios based on census data are therefore maximum ratios.[7] Since undocumented immigrants were probably recent arrivals, we assumed one uncounted immigrant of the same age, sex and birthplace for every two counted immigrants who entered the USA after 1969. This increased the English-speaking Caribbean adult female population by 23 percent, the Haitian population by 30 percent, and the "Other" population by 18 percent. We used this adjusted population to estimate *minimum* cancer incidence ratios, an arbitrary though "reasonable" maneuver that was necessary to assess the importance of unusually high ratios that might have programmatic implications.

Results

Incidence ratios for invasive cervical cancer were high for Haitian-born women, yet for carcinoma in situ, ratios were low for all three immigrant groups (Table 1). US-born women had a high incidence ratio for invasive cervical cancer but an average ratio for carcinoma in situ. For breast cancer all immigrant groups had low incidence ratios, whereas the ratio in US-born women indicated an incidence similar to the SEER rate. The SIRs cannot be compared to one another because they were not standardized to the same age distribution. When the population was adjusted for uncounted immigrants, all the SIRs were reduced, but relationships were essentially unchanged.

From the Departments of Obstetrics and Gynecology and of Preventive Medicine, SUNY-Brooklyn, and the Cancer Registry, New York State Department of Health. Address reprint requests to Rachel G. Fruchter, PhD, MPH, Associate Professor, Department of Ob/Gyn, Box 24, State University of New York-Health Science Center at Brooklyn, 450 Clarkson Avenue, Brooklyn, NY 11203. This paper, submitted to the Journal September 14, 1988, was revised and accepted for publication October 3, 1989.

TABLE 1—Standardized Incidence Ratios for Cancer of the Cervix and Breast by Birthplace among Black Women in Brooklyn, 1978–82

Cancer Site		Birthplace			
		USA	ESC*	Haiti	Other
A. Census Population					
Cervix (invasive)	N	296	78	36	26
	SIR	1.28	1.06	2.06	0.80
	95% CI	1.14, 1.43	0.84, 1.32	1.46, 2.85	0.52, 1.18
Cervix (in situ)	N	700	141	33	38
	SIR	0.96	0.64	0.48	0.38
	95% CI	0.89, 1.03	0.54, 0.75	0.33, 0.68	0.27, 0.52
Breast	N	754	206	45	40
	SIR	0.97	0.82	0.81	0.39
	95% CI	0.90, 1.04	0.71, 0.94	0.59, 1.08	0.28, 0.52
B. Adjusted Population					
Cervix (invasive)	SIR		0.90	1.62	0.72
	95% CI		0.72, 1.11	1.12, 2.25	0.47, 1.05
Cervix (in situ)	SIR		0.50	0.37	0.33
	95% CI		0.42, 0.60	0.26, 0.51	0.23, 0.43
Breast	SIR		0.71	0.64	0.33
	95% CI		0.62, 0.82	0.47, 0.86	0.23, 0.45

N = cases in five years (1978–82)
*English-speaking Caribbean

TABLE 2—Selected Characteristics of Black Women in Brooklyn by Birthplace

Characteristics	Brooklyn Women by Birthplace			National data
	USA	ESC	Haiti	
Fertility in women ages 15–24—Children per 1000 women	502	313	295	540
% Women over age 25 with education ≥ 12 grade	58.4	64.8	67.2	51.5
% Women ages 16 and over in labor force	45.8	67.0	63.2	53.3
% Persons with income below poverty	39.2	18.3	26.0	29.9

National data for Black women: US Census 1980.[12] Brooklyn data from Public Usage Microdata Sample for Brooklyn.

Selected characteristics of the four groups (excluding "other") are shown in Table 2. Immigrants had higher rates of high school completion and labor force participation and lower rates of poverty and fertility than US-born women in Brooklyn and Black women nationally.

Discussion

The incidence ratios reflect both the underlying prevalence of cervical neoplasia and the level of Pap smear screening. Factors consistently associated with a high prevalence of cervical neoplasia include low socioeconomic status (SES) and early sexual activity. These factors may account for the high incidence of invasive cervical cancer in US-born Black women in Brooklyn, also noted in Detroit, the major low-income, northern city in SEER.[8]

In the immigrant groups, the average to high rates of invasive cervical cancer combined with low rates of carcinoma in situ suggest low screening rates among all Black immigrants. Community and hospital studies of selected populations have consistently reported lower screening rates in immigrants than in US-born women.[1,2] A relatively low prevalence of cervical neoplasia in the Brooklyn immigrants may also be a possibility given their relatively high socioeconomic status and low early fertility (a proxy for sexual activity) reported in the census.

Cervical neoplasia among Haitian immigrants in Brooklyn has several unique features including: an unusually high incidence of invasive cancer (even when adjusted for a census undercount) with a high proportion in advanced stages,[1] and consistently lower screening rates than other Black women in community[2] and hospital populations.[1] Furthermore, two unpublished studies* suggest that Haitians have lower hysterectomy rates than other Black women in Brooklyn. First, in a community-site screening program, only 3 percent of 561 Haitian women had a hysterectomy compared to 17 percent of 378 English-speaking Caribbean immigrants and 14 percent of 319 US-born women. Second, among hospital inpatients with nongynecologic diagnoses undergoing routine cytology, none of 28 women with French names had a hysterectomy compared to 16 percent of 257 other Black women. In each study these differences held for all age groups, and the hysterectomy rates in the US-born and English-speaking Caribbean women were similar to rates in women in upstate New York.[9] Although conclusive evidence is lacking, we suggest that the uniquely high rate of invasive cervical cancer in Haitians may reflect a greater proportion of women with uteri at risk, low screening rates among Haitian immigrants in Brooklyn, and a severe lack of screening in deeply impoverished Haiti, where clinical and laboratory facilities are limited. A high rate of cervical neoplasia in Haitians has also been reported from Miami.[10]

The finding of an average incidence of breast cancer among US-born Black women in Brooklyn underlines the value of examining ethnic differences, since the overall rate in Black women would otherwise appear encouragingly low. The below-average incidence of breast cancer in both Haitian and English-speaking Caribbean immigrants is consistent with other studies which show persistently low rates of breast cancer in migrants from countries with low incidence.[11] However, limited exposure to clinical breast examination and mammography among immigrants might also contribute to low rates of detection of occult breast lesions. The demographic characteristics indicate that other risk factors associated with high rates of breast cancer—high SES and delayed pregnancy—cannot explain the low rates in immigrants.

These findings indicate the importance of evaluating cancer rates in different ethnic groups in heterogeneous communities, and have particular implications for cancer prevention activities in cities with Caribbean immigrant populations. The low rates of carcinoma in situ suggest that more basic education and cytologic screening are needed. The uniquely high rate of invasive cervical cancer in Haitians, combined with linguistic and other barriers to care, warrant special programs for Haitian women. Finally, the most intensive efforts to promote breast examination and mammography should probably be focused on US-born women.

ACKNOWLEDGMENTS

Supported by USPHS grant # 2SO7RR0540126 from the Biomedical Research Support Grant Program of The National Institutes of Health.

---

*Fruchter RG: Unpublished data.

Presented at the 115th Annual Meeting of the American Public Health Association, 1987.

REFERENCES

1. Fruchter RG, Remy JC, Boyce JG, Burnett WS: Cervical cancer in immigrant Caribbean women. Am J Public Health 1986; 76:797–799.
2. Fruchter RG, Wright C, Habenstreit B, Remy JC, Boyce JG, Imperato PJ: Screening for cervical and breast cancer among Caribbean immigrants. J Community Health 1985; 10:121–135.
3. NYS Cancer Registry: Cancer Incidence by County 1978–82. New York State Department of Health, Vol II, 1987; 305.
4. Horm JW, Asire AJ, Young JL, Pollack ES: Cancer Incidence and Mortality in the United States SEER 1973–81. US Dept Health Human Services. NIH Pub. No. 85-1837. Bethesda, MD: NIH, 1985.
5. Bailar JC, Ederer F: Significance factors for the ratio of a Poisson variable to its expectation. Biometrics 1964; 20:639–643.
6. NYC Department of City Planning: Caribbean Immigrants in New York City. 1985.
7. Woodrow K, Passell JS: Geographic distribution of undocumented immigrants: Estimates of undocumented aliens counted in the 1980 census by state. Int Migration Rev 1984; 18:642–671.
8. Bell SH, Schwartz AG, Swanson GM: Cancer Incidence in Metropolitan Detroit, 1979–81. Detroit, MI: Michigan Cancer Foundation, 1983.
9. Howe H: Age-specific hysterectomy and oophorectomy prevalence rates and the risks for cancer of the reproductive system. Am J Public Health 1984; 74:560–563.
10. Pitchenik AE, Becker DM, Hilsenbeck SG, Trapido EJ: Cancer among Haitians in Florida. Cancer 1987; 59:184–188.
11. Kelsey JL: A review of the epidemiology of human breast cancer. Epidemiol Rev 1979; 1:74–109.
12. US Census Bureau: General Social and Economic Characteristics: US Summary. PC80-1-C1, Tables 83, pp 21–23. Washington DC: Supt of Documents, 1983.

Ten-Year Changes in Birthweight Distributions of Black and White Infants, South Carolina

ROBERT M. MAYBERRY, PHD, AND ROBERT F. LEWIS, PHD

Abstract: The birthweight distributions of Black and White infants in South Carolina were compared for the paired-year periods 1975–76 and 1985–86. No discernible changes in birthweight distributions between the two time periods were observed especially among Black infants. The distributions among White infants reflected an overall improvement in birthweight most noticeably above 2500g. (*Am J Public Health* 1990; 80:724–726.)

Introduction

Birthweight is considered the most important predictor of infant mortality particularly for deaths occurring within the first 28 days of life.[1,2] While significant declines have occurred in infant mortality rates in the United States only slight declines in the incidence of low birthweight (<2500 grams) have been noted.[3,4] When noted, the declines were not observed for all segments of the population. Considerable racial differences persist; Black infants are twice as likely to be low weight at birth as White infants.[4]

South Carolina continues to have one of the highest infant mortality rates in the nation. Nevertheless, infant mortality rates and particularly neonatal mortality rates have declined substantially in the last decade. To determine whether improvement in birth weight has occurred in South Carolina, 1975–76 birthweight distributions of Black and White infants were compared with those of 1985–86.

Methods

Reported birthweights of infants born to mothers residing in South Carolina at the time of birth were obtained from public tapes of matched births and deaths provided by the Office of Vital Records and Public Health Statistics, South Carolina Department of Health and Environmental Control. Sex-specific birthweight distributions for Black infants and White infants were compared for the paired-year periods 1975–76 and 1985–86. Five-ounce weight classes were chosen to reflect the original measurements (ounces) and to adjust for propensity to report in quarter-pound units for infants weighing near average. Cumulative frequency distribution curves for each paired-year period and for each race-sex group were generated. Nonparametric procedures were used for statistical comparison.[5]

Results

There were no discernible changes in the birthweight distributions especially among Black infants and no statistically significant differences in birthweight distributions between the two time periods for any race-sex comparison (Figures 1 and 2). The shift toward larger infants was more obvious for White infants than for Black infants when comparing categorical values (Table 1). There was no change

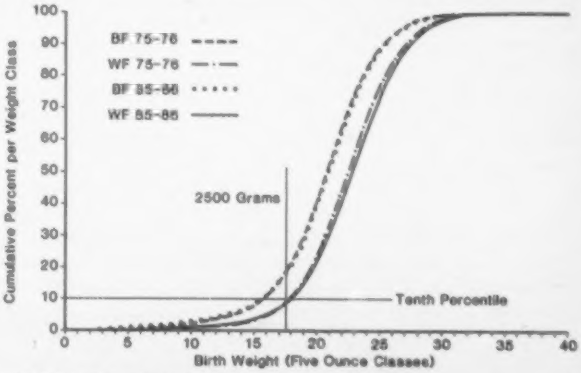


FIGURE 1—Cumulative Birthweight for Black and White Female Infants, South Carolina, 1975–76 vs 1985–86

From the Department of Epidemiology and Biostatistics, School of Public Health, University of South Carolina, Columbia. Address reprint requests to Robert M. Mayberry, PhD, Senior Epidemiologist for Minority Health, Centers for Disease Control (A-50), Atlanta, GA 30333. This paper, submitted to the Journal December 27, 1988, was revised and accepted for publication December 7, 1989.

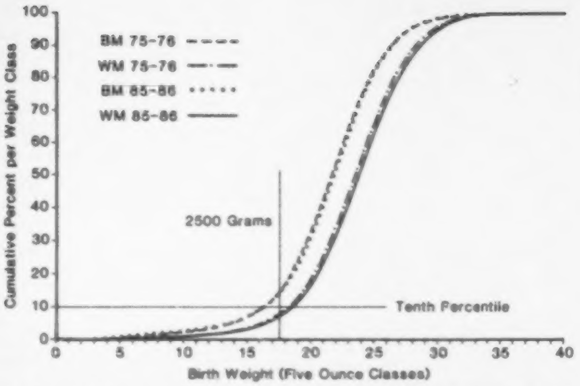


FIGURE 2—Cumulative Birthweight for Black and White Male Infants, South Carolina, 1975–76 vs 1985–86

in the proportion of Black or White infants weighing less than 2500g. Among White female and male infants, increases in birthweight were observed at the 10th, 50th, and 90th percentiles. Increases were observed among Black female infants only at the 90th percentile and among Black male infants only at the 50th percentile.

Changes were observed for certain maternal characteristics related to birthweight and/or infant mortality from 1975 to 1986 (Table 2). The proportion of teenagers giving birth declined from 32 percent to 23 percent among Blacks and from 19 percent to 12 percent among Whites accompanied by an increase in the proportion of women 30 years of age or older giving birth. A 16 percent difference (from 46 percent to 30 percent) in the proportion of mothers with less than a high school education was observed among Blacks with a 10 percent difference observed among Whites (from 31 percent to 21 percent). The proportion of unmarried mothers increased from 43 percent to 53 percent among Black women. There was little change in the proportion of expecting mothers seeking early prenatal care over the 10-year period.

TABLE 1—Summary Statistics, Birthweight Distributions of Black and White Infants for 1975–76 and 1985–86 Paired Years

Sex/Year	Number	Mean(g)	Standard Deviation(g)	LBW %	Percentiles(g) 10%	50%	90%
Females							
BF '75–76	18230	3029	600	14.4	2353	3090	3685
BF '85–86	19512	3036	618	14.2	2353	3090	3714
Difference		+7	+18	−0.2	0	0	+29
WR '75–76	27414	3300	564	6.7	2665	3317	3969
WF '85–86	30599	3335	579	6.7	2676	3374	3997
Difference		+35	+15	0.0	+11	+57	+28
Males							
BM '75–76	18684	3151	624	11.7	2438	3203	3855
BM '85–86	20008	3157	648	11.5	2438	3232	3856
Difference		+6	+24	−0.2	0	+29	+1
WM '75–76	29255	3424	602	6.0	2722	3458	4111
WM '85–86	32580	3465	605	5.4	2778	3487	4167
Difference		+41	+3	−0.6	+56	+29	+56

Discussion

It is clear that there has been no real improvement in the proportion of infants weighing less than 2500g between 1975 and 1986. Improvements in birthweight in this period are attributable to the segment of population producing less-than-average weight infants, but not so small as to qualify as low birthweight, i.e., <2500 g. The observations presented here are consistent with other reports showing only overall marginal and selective improvement in birthweights.[3]

Neonatal mortality rates in South Carolina declined by 32 percent among Blacks (from 17.2/1,000 live births to 11.7/1,000 live births) and by 39 percent among Whites (11.4/1,000 live births to 6.9/1,000 live births) from 1975 to

TABLE 2—Change in Selected Maternal Characteristics, 1975–76 to 1985–86

	Black			White		
	1975–76 (N = 37,189) %	1985–86 (N = 39,672) %	% difference	1975–76 (N = 57,548) %	1985–86 (N = 64,017) %	% difference
Age (years)						
<18	16	10	−6	7	4	−3
18–19	16	13	−3	12	8	−4
20–29	56	58	+2	67	64	−3
≥30	12	17	+5	14	23	+9
Education (years)						
<12	46	30	−16	31	21	−10
12	43	48	+5	42	39	−3
13–15	6	15	+9	15	20	+5
≥16	5	7	+2	12	19	+7
Marital Status						
Married	57	47	−10	95	92	−3
Unmarried	43	53	+10	5	8	+3
Prenatal Care Began (months)						
0	2	3	+1	1	1	0
1–3	52	53	+1	76	77	+1
4–6	37	34	−3	19	18	−1
7–9	8	10	+2	4	4	0

Totals may differ from 100% (or zero) because of rounding.

1986. (Office of Vital Records and Public Health Statistics, South Carolina Department of Health and Environmental Control, Columbia, SC.) Clearly the changes in neonatal mortality rates are not explained by changes in birthweight distributions. Low birthweight is an underlying contributor to neonatal mortality intricately related to a host of other biological, medical, sociodemographic, and behavioral factors. While many of these factors are vague, non-specific and non-operational, they are, in turn, related to specific, intervenable factors of prenatal care, nutritional intake, and smoking and alcohol consumption during pregnancy.[6] The explanation for the apparent paradox between significant declines in neonatal mortality rates and very marginal improvement in birthweight is not likely to be found in an examination of medical records and routinely collected vital statistics.

Finally, the absence of improvement in the proportion of low birthweight (LBW) infants over the time period 1975–86 should raise questions about either a flawed strategy of national health policy and/or inadequate application or utilization of support programs such as WIC (Women, Infants and Children supplemental feeding program), AFDC (Aid for Families with Dependent Children), and food stamps. Focusing on infant mortality as both a guide to operations and as a criterion for success in maternal and infant health programs is a suspect strategy. The effect of reducing the proportion of infants in the low birthweight tail of the distribution would certainly result in improvements in infant mortality rates, but not vice-versa. If the evidence of improvement among those whose infants weighing above the 2500g critical value is correctly interpreted, then there is more than a hint that the national strategy as it is applied in South Carolina should look beyond infant mortality. One approach would be to renew attention to conditions of life which are clearly known to influence maternal/familial well-being: hygiene, sanitation, crowding, nutrition, injury hazards, toxic substances, and unsafe conventions of behavior in the home and neighborhood.

REFERENCES

1. McCormick MC: The contribution of low birth weight to infant mortality and childhood morbidity. N Engl J Med 1985; 312:82–85.
2. IOM Committee to Study the Prevention of Low Birthweight: The significance of low birthweight. *In:* Institute of Medicine: Preventing Low Birthweight. Washington, DC: Academy Press, 1985; 21–45.
3. Kleinman JC, Kessel SS: Racial differences in low birthweight—trends and risk factors. N Engl J Med 1987; 317:749–753.
4. National Center for Health Statistics: Health, United States, 1985. DHHS Pub. No (PHS) 86-1232. Washington, DC: Govt Printing Office, 1985; 66.
5. Daniel WW: Biostatistics: A Foundation for Analysis in the Health Sciences. 4th Ed. New York: John Wiley and Sons, 1987; 583–636.
6. US Department of Health and Human Services: Report of the Secretary's Task Force on Black and Minority Health. Vol. VI: Infant Mortality and Low Birthweight. Washington, DC: Govt Printing Office, 1985; 35–85.

37

Unlicensed Midwifery Practice in Washington State

Susan J. Myers, LM, MPH, Patricia A. St. Clair, ScD, Stephen S. Gloyd, MD, MPH,
Philip Salzberg, PhD, and Joanne Myers-Ciecko

Abstract: We examined the role of unlicensed midwives in Washington State by questioning mothers of infants born out-of-hospital with an unlicensed person in attendance. Only a small proportion of the state's births (0.11 percent) were attended by unlicensed midwives. Unlicensed midwives attended 7 percent of home births, licensed midwives and certified nurse-midwives attended 69 percent. Mothers chose unlicensed midwives because they had religious beliefs in common, or because they were the only providers available who would attend a home birth. (*Am J Public Health* 1990; 80:726–728.)

Introduction

In 1981, the Washington State legislature passed a Midwifery Act which established standards for direct-entry (i.e., without prior nursing) midwifery education and practice. To become a licensed (direct-entry) midwife, the law specifies that applicants be graduates of a state accredited educational program wherein they receive at least three years of coursework and clinical experience. Only a handful of other states license direct-entry midwives. Washington's law is considered unique in the United States because its standards are consistent with those established by the International Confederation of Gynecology and Obstetrics and the International Confederation of Midwives.[1]

The Washington State Midwifery Act permits unlicensed midwives to practice as long as no fees are charged. In a recent review of the Act, the legislature added an amendment to allow unlicensed midwives with documented experience to challenge the licensing examination without completing the required coursework. The amended Act also stipulated that data be provided to determine the role of unlicensed practitioners in the provision of maternity services in the State of Washington. Accordingly, the present study was undertaken to determine the proportion of births that were attended by unlicensed midwives and other unlicensed attendants, and the reasons for mothers' choice of an unlicensed midwife.

Methods

Births which took place out-of-hospital (in a birth center, medical facility, home, or "other" setting) were selected from 1986 birth certificates (N = 1656). Those with an unlicensed person in attendance (mother, father, unlicensed midwife, other or unknown attendant) were identified (N = 423). Records

---

Address reprint requests to Susan J. Myers, LM, MPH, Seattle Midwifery School, 2524 16th Avenue South, Seattle, WA 98144. Ms. Myers is also with the Department of Health Services, University of Washington, which is also the affiliation of Dr. St. Clair and Dr. Gloyd. Dr. Salzberg is with Washington State Department of Licensing. Ms. Myers-Ciecko is with the Seattle Midwifery School. This paper, submitted to the Journal February 8, 1989, was revised and accepted for publication October 27, 1989.

TABLE 1—Birth Attendant Reported by Respondents Compared with Birth Attendant Recorded on Birth Certificate

Birth Attendant Recorded on Birth Certificate	Birth Attendant Reported by Respondents					
	Physician	Licensed Midwife/ Certified Nurse-Midwife	Father Mother	Unlicensed Midwife	Other	Total
Father/Mother	0	7	26	17	0	50
Unlicensed Midwife	0	9	0	12	1	22
Other/Unknown	47	11	15	38	23	134
Total	47	27	41	67	24	206

were checked by matching attendant's name on the birth certificate against state rosters of licensed providers.

Eight births were excluded because of a missing address or because the birth mother was not the infant's legal guardian. A questionnaire was mailed to the remaining 415 mothers, asking for verification of type of birth attendant and place of birth, whether they had planned an out-of-hospital birth, and the reasons for their choice of birth attendant. Follow-up telephone calls were made to non-respondents for whom telephone numbers could be found. A second questionnaire was sent to those who could not be reached by telephone.

Results

The response rate was 49.6 percent. Compared to respondents, non-respondents were more likely to be teenagers (11 vs 3 percent), other than White race (12 vs 5 percent), and unmarried (26 vs 7 percent). No differences were found in the proportion of mothers living in rural counties or having professional occupations.

The attendant category on the birth certificate agreed with the mother's response in only 30 percent of the cases (Table 1). Sixty-seven respondents said an unlicensed midwife attended their birth but only 12 birth certificates were correctly coded as such; 47 women said their birth attendant was a physician and 27 said the attendant was a licensed or certified nurse-midwife, for a combined total of 74 (36 percent) reporting that their birth attendant was actually licensed.

Table 2 shows the attendants and birthplaces for the 1656 out-of-hospital births with attendant classifications changed to reflect the 206 survey responses. The majority of these births occurred at home. Of the 76 births classified as having an unlicensed midwife attendant, 75 were home births. Of the 1,030 home births, licensed midwives accounted for the greatest proportion of attendants; unlicensed midwives attended only 7 percent.

TABLE 2—Out-of-Hospital Births by Type of Attendant and Place of Birth, Washington State, 1986

Birth Attendant	Out-of-Hospital Birthplace				
	Birth Center	Other Medical Facility	Home	Other	Total
Licensed Physician	155	97	78	8	338
Licensed Midwife	21	7	583	7	618
Certified Nurse-Midwife	267	2	131	0	400
Unlicensed Midwife	0	1	75	0	76
Mother/Father	0	1	65	2	68
Other	0	51	49	3	106
Unknown	0	0	49	1	50
Total	443	162	1030	21	1656

SOURCE: Washington State Vital Data, 1986, and 206 survey respondents

TABLE 3—Sociodemographic Characteristics of Mothers Who Gave Birth at Home By Attendant Type Washington State, 1986

Characteristics	Birth Attendant Types				
	% Unlicensed Midwife	% Licensed Midwife	% Nurse-Midwife	% Licensed Physician	% All Other*
Total	N: 75	N: 583	N: 131	N: 78	N: 163
Maternal age <20	1	2	2	9	7
Other than white race	4	4	3	26	13
Unmarried	15	11	5	29	18
Professional occupation (Father)	15	26	27	19	15
Professional occupation (Mother)	11	11	12	10	7
Rural county of residence	27	3	2	1	10

*Mother, Father, Other, Unknown

Source: Washington State Vital Data, 1986, and 206 survey respondents

Licensed midwives and certified nurse-midwives were more likely than unlicensed midwives to serve urban, married professionals (Table 3), while unlicensed midwives attended mothers residing in rural counties.

Only 13 percent of respondents said their out-of-hospital birth was unplanned. All such respondents were attended by a mother, father, friend, or other person.

Reasons for the choice of an unlicensed midwife include: "I could not find a licensed birth attendant in my community willing to attend an out-of-hospital birth" (31 percent), and "A major factor in my decision was to have someone to care for me with religious beliefs similar to my own" (31 percent).

Most respondents attended by unlicensed midwives were satisfied with the care they received, although 25 percent reported that they would have preferred a licensed midwife or doctor. Sixty-seven percent said their unlicensed midwife charged a fee for her services.

Discussion

These results suggest that unlicensed midwives attend a small proportion (0.11 percent) of births in Washington State. However, for several reasons, our data may underestimate the true proportion of unlicensed midwife-attended births. If it is assumed that all unlicensed attendants of out-of-hospital births were actually unlicensed midwives, approximately 18 percent of the 1,656 out-of-hospital births would have been attended by unlicensed midwives, 0.43 percent of the state's total births. Even at this upper end of the range of plausible estimates, the proportion of unlicensed midwife-attended births is still quite small.

If the mother's questionnaire response is correct, the extent of attendant misclassification on birth certificates was surprising. Although we expected to find unlicensed midwife attendants concealed within the other unlicensed categories, there were also numerous instances of births attended by licensed providers that had been attributed to unlicensed attendants on the birth certificate. In these cases, birth certificates were either inaccurately completed or coded incorrectly.

Apparently many respondents chose an unlicensed midwife because they were unable to locate a licensed provider who would attend an out-of-hospital birth. This is consistent with the finding that women attended by unlicensed midwives are more likely to reside in rural counties than their counterparts attended by licensed midwives or certified nurse-midwives.

Shared religious beliefs was another reason cited for the choice of birth attendant. It was later learned that some unlicensed midwives regard their practice as a ministry to serve fellow churchwomen.

That two-thirds of mothers attended by unlicensed midwives paid a fee for service suggests that in most cases this was a professional relationship, not simply one friend helping another. Technically, unlicensed midwives who charge a fee are practicing illegally. However, because they are responding to the needs of particular groups of women, they are likely to continue to practice in spite of legal prohibitions. Legislation prohibiting unlicensed midwives from practicing is rarely enforceable.[2] Indeed, the real effect of such legislation has been to force unlicensed midwives to work in secret or to prevent them from seeking education and training.[2,3] Providing an avenue to legal midwifery practice may be a more appropriate course of action so long as competence, equivalent to other licensed providers, can be demonstrated.

REFERENCES

1. Joint Study Group of the International Federation of Gynecology and Obstetrics and the International Confederation of Midwives: Maternity Care in the World. Hampshire, England: C.M. Printing Services, 1976.
2. Owen M: Laws and policies affecting the training and practice of traditional birth attendants. Int Digest Health Legis 1983; 34:441–475.
3. World Health Organization: Report of the Consultation on Approaches for Policy Development for Traditional Health Practitioners, Including Traditional Birth Attendants. Geneva: WHO, 1985; 18.

Occupational Physical Activity and other Risk Factors for Preterm Birth among US Army Primigravidas

GILBERT RAMIREZ, DRPH, RICHARD M. GRIMES, PHD, JOHN F. ANNEGERS, PHD,
BARRY R. DAVIS, MD, PHD, AND CARL H. SLATER, MD

Abstract: We examined the relation of occupational physical activity to the risk of preterm birth among US Army active-duty primigravidas between 1981 and 1984 using 604 cases (preterm deliveries; ≤ 37 weeks gestation) and 6,070 controls (term and post-term deliveries). Women employed in the highest physical activity levels had increased odds of preterm delivery ranging from 1.69 to 1.75. The relation was not changed by adjustment for the effects of age, marital status, socioeconomic status, or education. Missing data suggest cautious interpretation. (*Am J Public Health* 1990; 80:728–729).

Introduction

Relatively few studies have examined the relation between physical activity on the job and the risk of preterm birth, and the findings have been mixed. In a French case-control study, risk of preterm birth was related to the number of sources of occupational fatigue and with a work-week longer than 40 hours.[1] This finding was not confirmed in a case-control study in New Haven, Connecticut;[2] decreased risk of preterm birth was seen among women participating in sports or physical fitness exercises during pregnancy.

The population of US Army active-duty gravidas presents an unusual opportunity to study the relation between preterm birth and occupational physical activity because of its large size and homogeneity, and because US Army military occupational specialties (MOS) had undergone physical demand assessments.

Methods

In 1977, entry level specialties for enlisted personnel were categorized by maximum upper body strength requirements as required for "combat conditions" performance (see Appendix).[3] This physical activity measure was not a specific measure of actual physical activity during pregnancy.

Data for this study were obtained from Patient Administration Systems and Biostatistics Activity, Fort Sam Houston, Texas for length of gestation (from maternal and infant inpatient records) and from US Army Military Personnel Center, Alexandria, Virginia for physical activity and other demographic data (from fiscal year personnel files).

Each subject was assigned to her MOS physical activity level during pregnancy using the fiscal year corresponding to her mid-gestation date. US Army policy forbids the reassignment of women during pregnancy.

The population of US Army active-duty gravidas delivering at US Army medical treatment facilities numbered 22,450 during the four-year period 1981–84. This period was chosen because of differences in coding schema for inpatient records prior to 1981; data for deliveries after 1984 were incomplete at the time of this study. Eligibility criteria for the study sample included primiparity, singleton delivery, and enlisted paygrade status. Officer occupations had not been assessed for physical demand requirements.

Cases were defined as preterm deliveries (≤ 37 weeks gestation); controls included term and post-term deliveries.

Address reprint requests to Gilbert Ramirez, DrPH, Assistant Professor, Graduate Program in Allied Health Research, Lavaca Hall West, Southwest Texas State University, San Marcos, TX 78666-4616. Dr. Ramirez is a Major (Ret), US Army. All co-authors are from the University of Texas Health Science Center, School of Public Health, Houston, Texas. This paper, submitted to the Journal September 7, 1988, was revised and accepted for publication August 25, 1989.

Risk factors studied included age, race, pay grade, length of military service, marital status, education, occupational aptitude score, job skill level, and type of military unit. The latter two variables were included to control for the initial focus of the MOS physical demand analysis: "entry level skills in a combat environment." Military duties performed in a field unit closely resemble how they would be performed in a combat environment. The occupational aptitude score was included since it is also used to determine MOS eligibility.

Odds ratios were computed for each separate risk factor. The dose-response relation between occupational physical activity and preterm birth was assessed with the Mantel test for trend.[4] We also fitted a logistic regression model and estimated population attributable risk.[5]

Results

Of the 22,450 US Army active-duty deliveries between 1981–84, 20,684 were to enlisted women. Of these, 20,218 were single live births. Using the mothers' Social Security Numbers (SSN), 12,911 of the 20,218 (64 percent) maternal inpatient records could be matched to infants' inpatient records. Seventy-two percent (9,262) of these matched records met the primiparity criterion. Using the maternal SSN, 7,821 were matched with personnel records. Of these, 1,147 records could not be used due to MOS data entry errors. The remaining sample of 6,674 included 604 cases and 6,070 controls.

Odds ratios for each risk factor are presented in Table 1. Risk factors for preterm birth included the two highest physical activity categories, age less than 20 years, other than White race, pay grades E1 to E3, less than two years' total military service, a never-married status, and an occupational aptitude score less than 100. The level of physical activity was positively related to risk. Condensing the five-category physical demand model to three categories, we found only minor changes in odds ratio estimates and chi-square values by controlling for other risk factors one at a time using a stratified Mantel trend test.*

TABLE 1—Estimates of Relative Risk for Preterm Birth

Risk Factors	Number	Percent Preterm	Odds Ratio	95% CI
Physical demand				
Low	429	6.1	1.0	
Medium	1949	8.5	1.43	0.92,2.25
Moderately Heavy	940	8.4	1.42	0.88,2.31
Heavy	1825	9.8	1.69	1.08,2.64
Very Heavy	1531	10.1	1.75	1.12,2.75
(p-value, trend: 0.0056)				
Age (years)				
≥20	5925	8.5	1.0	
<20	749	13.8	1.73	1.37,2.18
Race				
White	2927	7.7	1.0	
Other	3747	10.1	1.34	1.12,1.60
Paygrade				
E4–E8	4361	7.6	1.0	
E1–E3	2313	11.8	1.64	1.38,1.95
Years of service				
≥2	4132	7.9	1.0	
<2	2542	10.9	1.42	1.20,1.69
Marital status*				
Ever Married	3434	8.1	1.0	
Never Married	3239	10.0	1.26	1.06,1.50
Aptitude Score*				
≥100	3871	7.6	1.0	
<100	2583	11.1	1.50	1.26,1.79
Years of education*				
>12	839	7.9	1.0	
≤12	5832	9.2	1.19	0.90,1.57
Job skill level				
Levels 2–5	1437	8.1	1.0	
Entry	5237	9.3	1.17	0.94,1.45
Type of unit				
Garrison	2430	9.2	1.0	
Field	4244	9.0	1.02	0.86,1.22

*Missing data

The analysis of a multifactorial model using logistic regression resulted in three significant predictors of preterm birth: physical activity, race, and military pay grade. The combined risk of a preterm birth for all three factors ranged from 3.3 for those employed in "heavy" physical activity to 3.6 for those employed in "very heavy" physical activity. The population attributable risk for the risk factor occupational physical activity was estimated as 17.5 percent when adjusted for pay grade and race.*

Discussion

The results of this study should be interpreted with caution for several reasons. First, a considerable amount of information was lost when maternal inpatient records were not matched to their infants' inpatient records or their personnel records. Approximately one-fifth of the lost information was from the births occurring in civilian hospitals which do not forward information pertaining to the infant to the nearest military facility. The remaining four-fifths of data were lost because social security numbers did not match. Prior to 1985, there was no uniform policy regarding the entry of social security number on an infant's inpatient record when both parents were active-duty. The impact of the lost information may be small, however, since the dependent variable, length of gestation, and the primary independent variable, physical activity, were obtained from two independent sources, and there is no reason to suspect any systematic bias with regard to these two variables.

A greater concern is that information was not available to this study on potential confounders such as nutrition practices, smoking habits, alcohol consumption, and pregnancy complications.

Other studies have shown activity-duty gravidas to be at high risk for adverse reproductive outcomes, particularly when compared to wives of active duty males.[6,7] Active-duty gravidas are required to continue work until delivery; although military physicians provide recommendations to the pregnant soldier's commander regarding restrictions, these are often general in nature and rarely address physical activity beyond prohibiting participation in regular unit physical fitness programs.

The strength of this study was the consistency of the observed relationship between occupational physical activity and the risk of preterm birth even after controlling for risk factors such as age, race, and socioeconomic status. Additional research in this area seems called for.

ACKNOWLEDGMENTS

The views presented here are of the authors and do not purport to reflect the position of the US Department of the Army or the Department of Defense.

---

*Data available upon request to author.

APPENDIX

US Army Physical Demand Categories

Category	Maximum Lift Criteria (lbs)* Frequent or Occasional	Constant
Light	20	10
Medium	50	25
Moderately heavy	80	40
Heavy	100	50
Very heavy	>100	>50

*Occasional: <20% of the time
Frequent: >20% but < 80% of the time
Constant: >80% of the time

REFERENCES

1. Mamelle N, Laumon B, Lazar P: Prematurity and occupational activity during pregnancy. Am J Epidemol 1984; 119:309–322.
2. Berkowitz GS, Kelsey JL, Holford TR, Berkowitz RL: Physical activity and the risk of spontaneous preterm delivery. J Reprod Med 1983; 28:581–588.
3. Women in the Army Policy Review Group. Women in the Army policy review. Washington, DC: Department of the Army, 1982.
4. Schlesselman JJ: Case-control studies. Design, conduct, analysis. New York: Oxford University Press, 1982; 200–206.
5. Kleinbaum DG, Kupper LL, Morgenstern H: Epidemiologic Research. Principles and quantitative methods. London: Lifetime Learning Publications, 1982; 160–164, 434–435.
6. Fox ME, Harris RE, Brekken AL: The active-duty military pregnancy: A new high-risk category. Am J Obstet Gynecol 1977; 129:705–707.
7. Hauth JC, Gilstrap III LC, Brekken AL: The effect of 17α-hydroxyprogesterone caproate on pregnancy outcome in an active-duty military population. Am J Obstet Gynecol 1983; 146:187–190.

Pawtucket Heart Health Program Point-of-Purchase Nutrition Education Program in Supermarkets

Mary K. Hunt, MPH, RD, R. Craig Lefebvre, PhD, Mary Lynne Hixson, MA, RD,
Stephen W. Banspach, PhD, Annlouise R. Assaf, PhD, and Richard A. Carleton, MD

Abstract: Point-of-purchase nutrition education in supermarkets is one intervention strategy of the Pawtucket Heart Health Program, a community cardiovascular disease prevention program in Pawtucket, Rhode Island.

Using consumer intercept interviews, awareness of shelf labels and their effect on purchase behavior have been continuously evaluated. Between 1984 and 1988, the percent of shoppers who could identify correct labels increased from 11 percent to 24 percent (95% confidence intervals of difference: 7,17). The percent who reported they were encouraged to purchase the identified foods increased from 36 percent to 54 percent (95% CI of difference: 5,41). (*Am J Public Health* 1990; 80:730–731.)

Introduction

Because 57 percent of the adult population has blood cholesterol levels that put them at moderate or high risk for cardiovascular disease (CVD),[1] many Americans need to change their dietary intake of saturated fat and cholesterol to reduce their blood cholesterol level and CVD risk.

To reach this large number of people, Blackburn and Kottke delineate the need for population strategies.[2,3] The Pawtucket Heart Health Program, a comprehensive community heart disease prevention study,[4–7] developed the "Four Heart Program," a point-of-purchase (POP) nutrition education program in supermarkets and restaurants, as one of its population strategies. We report the results of a study of consumer awareness of the supermarket program and its influence on self-reported purchase behavior.

Methods

The Four Heart supermarket program was started in three supermarkets owned by two different companies and one small family-owned market in 1983–84. The name, Four Heart, represents foods that are tasty and contain less fat, cholesterol, and sodium. The basis of the program is the placement of brand-specific shelf labels next to the unit price tags of qualifying foods. Original multi-colored shelf labels that identified products low in sodium, fat, and/or calories were replaced in 1986 with labels of uniform color with the messages: "low-fat", "low-sodium", "low-fat, low-sodium", and "fat ratio OK." Collateral and support materials include signs with health messages in areas where shelf labels cannot be applied; "Look for the Labels" posters with lucite holders for brochures; free recipe cards; periodic promotions such as contests and blood pressure and cholesterol screening, counseling and referral events (SCORES); and training of store and department managers and of lay volunteers as program monitors.

Criteria used to determine the eligibility of foods for these labels were adapted from the Minnesota Heart Health Program[8] and Food and Drug Administration (FDA) food labeling regulations and recommendations[9,10]

Whereas FDA-sponsored descriptive programs labeled all products in the store that met their nutrient criteria,[11,12] we were interested in studying a more prescriptive program. Only those foods included in meal pattern recommendations based on the US Dietary Guidelines for Americans[13] are labeled.

Consumer intercept interviews were conducted at approximately yearly intervals over four years in two supermarkets from two chains. Interviews were administered in a similar manner across all stores and time periods.

From the Division of Health Education, Memorial Hospital of Rhode Island, Pawtucket, RI. Address reprint requests to Mary Lynne Hixson, MA, RD, Pawtucket Heart Health Program, Memorial Hospital of RI, 111 Brewster St., Pawtucket, RI 02860. Additional affiliations of the co-authors are: Department of Community Health, Brown University, Providence, RI (Lefebvre, Banspach, Assaf); and the Division of Cardiology, Memorial Hospital of Rhode Island, and Department of Medicine, Brown University (Dr. Carleton). This paper, submitted to the Journal August 8, 1989, was revised and accepted for publication December 20, 1989.

Participants

Individuals exiting a store who were judged to be at least 18 years old were eligible for the interview. Once selected, an individual exiting the store was asked if s/he had purchased something in the store. Those who indicated that they had made a purchase were interviewed.

Procedure

Shoppers were selected for interviews based on a time sampling procedure. Using a stop watch, the interviewer selected a potential respondent by approaching the first age-eligible shopper to exit the store at five-minute intervals.

Instrument

Data on frequency of shopping were obtained. To assess awareness of shelf labels, a placard containing four sets of labels, three of which were bogus sets, was shown to the respondent. The shopper was asked if s/he saw any of these labels inside the store. If the answer was yes, s/he was asked to identify the set seen. Those shoppers who reported seeing the labels actually on the shelves were counted to assess the influence of the labels on purchase behavior.

Results

Awareness of Labeled Items

Interviews with 1,807 shoppers were conducted over the four-year period. Fifteen percent more shoppers reported seeing any labels on the store shelves between 1984 to 1988 (Table 1).

Awareness was related to gender; 32 percent of female shoppers versus 24 percent of male shoppers reported seeing one of the labels shown to them (95% CI = 4,12). Thirteen percent more shoppers were able to identify the correct label between 1984 and 1988 (Table 1). Female shoppers identified the correct labels 23 percent of the time compared with 12 percent for males (95% CI = 7,15).

Influence of Shelf Labels on Purchase Behavior

Encouragement to purchase labeled foods was reported more frequently in 1988 than in 1984 among shoppers who correctly identified Four Heart labels (Table 1). Encouragement to purchase labeled products did not differ appreciably between male and female shoppers.

TABLE 1—Consumer Awareness and Effect of Food Product Shelf Labeling

	Total Surveyed	Yes[a]	Correct[b] Label	Encouraged[c] Purchase
	n	n %	n %	n %
1984	355	72 (20)	39 (11)	14 (36)
1986	454	104 (23)	70 (15)	36 (51)
1987	418	150 (36)	98 (23)	58 (59)
1988	531	185 (35)	129 (24)	70 (54)
		95% confidence intervals of difference		
'88 vs '86		6, 18	4, 14	
'88 vs '84		9, 21	8, 18	7, 35
'87 vs '86		7, 19	3, 13	
'87 vs '84		10, 22	7, 17	5, 41

[a] Shoppers reporting having seen any label shown of total surveyed
[b] Shoppers correctly identifying the "4-Heart labels" of total surveyed
[c] Shoppers stating that label encouraged purchase of those who saw correct label

Discussion

The supermarket program reached large numbers of people with low fat, low sodium eating pattern messages over a four-year period of time. In order to quantify this reach, we applied findings from our study to industry averages obtained from the Food Marketing Institute, Washington, DC (personal communication). In 1987, the mean weekly store sales in US supermarkets was $184,210 with an average transaction of $16.30. This translates to 11,301 transactions per store per week.

Based on our interviews, customers shopped an average of 2.3 times a week. Therefore, we estimate that the participating supermarkets served 4,913 customers a week.

Projecting our findings from these averages, by 1988 each week an estimated 1,179 customers recognized Four-Heart shelf labels while shopping and 636 of them were encouraged to purchase labeled products. It would be difficult and expensive to achieve this exposure to health messages and change in purchase behavior with one-to-one or small group methods of nutrition education.

Although a larger percentage of females than males identified the correct labels, approximately one-half of both males and females reported that they were encouraged to purchase the labeled foods. This is of particular interest because males have lower participation rates in traditional nutrition education programs than females[14,15] and are at higher risk for CVD—one in three will have clinical signs of CVD before the age of 60.[16]

Two important caveats must be given to these results. The first is that this program evaluation occurred within the context of a large, broad-based intervention effort to raise awareness of, and stimulate changes in, CVD risk behaviors. Secondly, other secular trends such as the increasing media coverage of cholesterol-related topics, and the efforts of the National Cholesterol Education Program and American Heart Association in this area, may also have added to the observed increases.

Yet, our data do support the findings of other studies which have demonstrated that point of purchase nutrition education in supermarkets is feasible and effective in increasing consumer awareness of nutrition messages over time[11,17,18] and in exerting a positive influence on self-reported purchase behavior.[18] Because of the ambiguous results of studies of actual consumer purchase behavior,[11,12,17,19–22] further controlled studies of this intervention in supermarkets need to be undertaken.

ACKNOWLEDGMENTS

The authors thank the management and staff of Pawtucket area Almac's Inc. and Star Market Company for their cooperation and support of the Four Heart Program and its evaluation. This research was supported by Grant HL23629 from the National Heart, Lung and Blood Institute, US Department of Health and Human Services.

REFERENCES

1. Sempos C, Fulwood R, Haines C, *et al:* The prevalence of high blood cholesterol levels among adults in the United States. JAMA 1989; 262:45–52.
2. Blackburn H: Research and demonstration projects in community cardiovascular disease prevention. J Public Health Policy 1983; 4:398–421.
3. Kottke TE, Puska P, Salonen JT, Tuomilehto J, Nissinen A: Projected effects of high risk versus population based prevention strategies in coronary heart disease. Am J Epidemiol 1985; 121:697–703.

4. Lefebvre RC, Lasater TM, Carleton RA, Peterson G: Theory and delivery of health programming in the community: The Pawtucket Heart Health Program. Prev Med 1987; 16:80–95.
5. Carleton RA, Lasater TM, Assaf A, Lefebvre RC, McKinlay S: The Pawtucket Heart Health Program: I. An experiment in population-based disease prevention. RI Med J 1987; 71:533–538.
6. Lasater TM, Lefebvre RC, Carleton RA: The Pawtucket Heart Health Program: IV. Community level programming for heart health. RI Med J 1987; 71:31–34.
7. Assaf AR, Banspach SW, Lasater TM, McKinlay SM, Carleton RA: The Pawtucket Heart Health Program: II. Evaluation strategies. RI Med J 1987; 71:541–546.
8. Mullis RM, Snyder P, Hunt MK: Developing nutrient criteria for food specific dietary guidelines for the general public. J Am Dietet Assoc (in press).
9. US Department of Health and Human Services, Food and Drug Administration. Food Labeling; declaration of sodium content of foods and label claims for foods on the basis of sodium content; final rule, Rules and Regulations. Federal Register April 18, 1984; 49:155110–35.
10. Pennington JT, Wisniowski LA, Logan GB: In-store nutrition information programs. J Nutr Educ 1988; 20:5–10.
11. Ernst ND, Wu M, Frommer P, *et al:* Nutrition education at the point of purchase: the foods for health project evaluated. Prev Med 1986; 15:60–73.
12. Levy AS, Matthews O, Stephenson M, Tenney JE, Schucker RE: The impact of a nutrition information program on food purchases. J Public Policy Market 1985;4:1–13.
13. US Departments of Agriculture and of Health and Human Services. Food 1, Home and Garden Bulletin No. 228. Washington, DC: Govt Printing Office, 1979.
14. Spilman MA: Gender differences in worksite health promotion activities. Soc Sci Med 1988; 26:525–535.
15. Lefebvre RC, Harden EA, Rakowski W, Lasater TM, Carleton RA: Characteristics of participants in community health promotion programs: Four-year results. Am J Public Health 1987; 77:1342–1344.
16. Atherosclerosis Study Group: Optimal resources for primary prevention of atherosclerotic diseases. Circulation 1984; 70:157A–205A.
17. Russo JE, Staelin R, Nolan CA, Russell GJ, Metcalf BL: Nutrition information in the supermarket. Consum Res 1986; 13:48–70.
18. Mullis RM, Hunt MK, Foster M, *et al:* The Shop Smart for Your Heart Grocery Program. J Nutr Educ 1987; 19:225–228.
19. Mayer JA, Dubbert PM, Elder JP: Promoting nutrition at the point of choice: A review. Health Educ Q 1989; 16:31–43.
20. Muller TE: Structural information factors which stimulate the use of nutrition information: A field experiment. Market Res 1985; 22:143–157.
21. Soriano E, Dozier DM: Selling nutrition and heart-healthy diet behavior at the point-of-purchase. J Appl Nutr 1978; 30:56–65.
22. Jeffery RW, Pirie PL, Rosenthal BS, Gerber WM, Murray DM: Nutrition education in supermarkets: An unsuccessful attempt to influence knowledge and product sales. J Behav Med 1982; 5:189–200.

Infant Feeding Practices: An Evaluation of the Impact of a Health Education Course

ILANA SHOHAM-YAKUBOVICH, MPH, PHD, JOSEPH S. PLISKIN, PHD, AND DANIEL CARR, MS

Abstract: We assessed the impact of a health education course on infant feeding practices in the West Bank territories by comparing mothers who had attended the course (n = 102) with mothers not exposed to the course (n = 133). After adjustment for child's age, maternal age and education, parity, and birth site, course participants were more likely than non-participants to breastfeed, as well as to start supplementation by semi-solid foods at the recommended time. (*Am J Public Health* 1990; 80:732–733.)

Introduction

A comprehensive project of health education for mothers of young children in the villages of the West Bank territories, a population of low socioeconomic status, was designed as part of a food distribution activity.[1,2] Prior assessment of needs and problems of the local population indicated that a growing number of the mothers wean their children too soon, and introduce semi-solid foods either too early or too late. One of the main objectives of the course was to change these feeding practices. The course was taught in small groups by specially trained local instructors who adapted teaching materials developed for the course to the specific questions and issues raised by their groups. The course focused on nutrition, hygiene, child development, and first aid. The process of course development, design, methods, and content were described elsewhere.[1,2]

From 1985 to 1987, the program offered 972 courses in 152 villages (about one-third of the 443 villages in the West Bank) and in five cities throughout the West Bank. Participation of villages in the program was voluntary. In participating villages, most mothers of young children joined the course. Participating mothers received a small supply of basic food commodities. Overall, 19,984 mothers participated in these courses.

The present paper assesses the impact of the course on infant feeding practices, by comparing a sample of mothers who had attended the health education classes with a sample of mothers who had not been exposed to the course.

Methods

A survey was conducted in 92 villages throughout the West Bank, 46 which participated in the program, and 46 which had not participated. The sample of villages was randomly selected in proportion to the population distribution in six districts. Approximately six mothers in each village were interviewed. In experimental villages, the respondents were randomly selected from the list of course participants. In the comparison villages, respondents were selected in most cases from among the mothers whose children were attending school at the time of the survey. Structured interviews were conducted between August and December 1987 by female field workers experienced in village habits and familiar with local customs and culture. The present analysis includes 235 mothers whose youngest child was one year or less. Of these, 102 attended the course and 133 mothers did not attend the course. Statistical analysis

---

Address reprint requests to Ilana Shoham-Yakubovich, MPH, PhD, Epidemiology and Health Services Evaluation Unit, University Center for Health Sciences, Ben-Gurion University of the Negev, P.O. Box 653, Beer Sheva, Israel. Dr. Pliskin is with the Department of Industrial Engineering and Management at the University; Mr. Carr is with Catholic Relief Services, Baltimore, Maryland. This paper, submitted to the Journal June 5, 1989, was revised and accepted for publication December 8, 1989.

---

includes comparisons of crude and age-adjusted proportions. Confidence intervals (CI) for differences of crude and standardized proportions were calculated by methods suggested by Rothman.[3] Mantel-Haenszel chi square was used to assess statistical significance of stratified analysis.[4] Multivariate logistic regression was performed to assess the effect of the health education course on feeding practices, controlling for other variables.[3]

Results

There were no differences of consequence in maternal age, number of children, and maternal level of formal education between the two groups (Table 1), but 51.1 percent of the course participants delivered at home assisted by a local midwife (Daya) or by a female relative, while only 35 percent in the comparison group delivered at home. All other women delivered in a clinic or a hospital.

Proportions of breastfeeding mothers were higher in the course group than in the comparison group, irrespective of child's age (Table 2). Introduction of semi-solid foods at 4–6 months (the recommended pattern) was more prevalent in the course group than in the comparison group (76 and 57 percent, respectively; 95 percent CI for the 19 percent difference = 7.1–30.9). Starting semi-solid foods at age 6 months was a more common practice in the comparison group than in the course group (16 and 1 percent, respectively).

Women who gave birth at home were more likely to start breastfeeding than women delivering in the hospital (97 vs. 74 percent), and to be still breastfeeding at 10–12 months (58 percent vs. 42 percent). Comparing rates of breastfeeders (adjusted for child's age) demonstrates that course participants were more likely to breastfeed, regardless of site of delivery (home vs. hospital or clinic). The difference, however, significantly exceeds 0 only among mothers delivering in the hospital (Table 3).

After controlling for child's age, site of delivery, maternal age, level of education, and parity, course participation continued to be associated with breastfeeding at the time of interview, as well as with starting supplementation with semi-solid foods at age 4 months (Table 4). Breastfeeding was also associated with child's age and home birth. The recommended pattern of supplementation with semi-solid foods, was associated with course participation, maternal education, and number of children (Table 4).

TABLE 1—Comparison of Socio-Demographic Characteristics of Mothers to Children 0–1 Years Old in the Experimental Group and the Comparison Group

	Experimental Group		Comparison Group	
	$\bar{X}$	SD	$\bar{X}$	SD
Age	27.4	(6.6)	28.5	(6.2)
No. of children	4.6	(2.6)	4.9	(2.7)
Years of schooling	6.1	(3.4)	5.2	(4.2)
% of women delivering youngest child in hospital or clinic	34.6		51.1*	

*Difference 16.5% (95% CI = 3.1, 29.9).

TABLE 2—Percent of Women Breastfeeding by Child's Age and Course Participation

	Experimental Group		Comparison Group	
Child's Age (mos)	%	n	%	n
0–3	100.0	(25)	78.4	(29)
4–6	84.0	(21)	65.7	(23)
7–9	70.8	(17)	62.5	(20)
10–12	64.3	(18)	37.9	(11)

Mantel-Haenszel $x^2 = 6.802$, $p < .01$ (3).

TABLE 3—Percent of Women Breastfeeding at the Time of Interview, Adjusted for Child's Age,[a] by Site of Delivery, and Course Participation

	Home Delivery		Hospital Delivery	
	%	n	%	n
Experimental Group	79.1	(66)	83.1	(36)
Comparison Group	68.7	(65)	54.3	(68)
Difference	10.4		28.8	
95% CI[b]	(−3.5, 24.3)		(13.9, 43.7)	

[a] Rates were adjusted for child's age by the direct method, using the age group distribution of the entire sample as the standard population.

[b] 95% confidence intervals for standardized rates.[3]

TABLE 4—Results from Multiple Logistic Regression Analysis Predicting Infant's Feeding Mode

	Breastfeeding at Present (Yes/No)		Introducing Semi-Solid Food at 4–6 Months* (Yes/No)	
Variables	Odds Ratio	95% CI	Odds Ratio	95% CI
Course participation (yes/no)	2.81	2.03, 3.88	2.63	1.89, 3.70
Home delivery (yes/no)	1.67	1.23, 2.28	1.37	0.98, 1.93
Mother's age[a]	1.10	0.89, 1.35	0.81	0.64, 1.02
Mother's education[b]	1.10	0.94, 1.28	1.44	1.21, 1.71
N of children[c]	1.00	0.82, 1.22	1.32	1.05, 1.65
Child's age[d]	1.85	1.61, 2.13	1.03	0.85, 1.26

[a] Age: (<25, 25–29, 30–35, >35)

[b] Years of schooling: (0, 1–5, 6–8, >8)

[c] Number of children: (1–2, 3–4, 5–6, >6)

[d] Months: (0–3*, 4–6, 7–9, 10–12)

*Only mothers whose youngest child was 4 months or older at the time of the interview were included in this part of the analysis.

Discussion

The pattern of findings is consistent with the conclusion that the course was successful in influencing mothers' feeding habits. The results, however, could have been influenced by several biases inherent in the study design. Since our study is cross-sectional, and since the course was not randomly assigned, it is possible that health education project villages differed from the comparison villages in aspects relevant to child feeding practices for which no information was available. However, stratification on site of last delivery and multivariate adjustment for maternal age and education, site of delivery, number of children, and child's age suggest that course participation had a unique contribution to feeding behavior.

Another possibility is that women in the health education group report, but do not behave, differently than the comparison group. Even if some reporting bias does exist, the mere knowledge in itself about appropriate practices is a worthwhile benefit of the course.

Our findings are consistent with a previous study showing a beneficial effect of special class on breastfeeding among mothers of low socioeconomic status.[5] These findings are encouraging in light of the trend observed among mothers of low socioeconomic groups,[6,7] and mothers in third world countries of declining rates of breastfeeding.[8]

REFERENCES

1. Carr D: Village mothers on the West Bank learn about health. World Health Forum 1988; 9:245–249.
2. Shoham-Yakubovich I, Pliskin JS, Carr D: The impact of a health education course on maternal knowledge: A comparative study in a low socioeconomic rural region. Health Educ Q 1990 (in press).
3. Rothman KJ: Modern Epidemiology. Boston: Little, Brown & Co, 1986.
4. Mantel N, Haenszel W: Statistical aspects of the analysis of data from retrospective studies of disease. JNCI 1959; 22:719–748.
5. Young SA, Kaufman M: Promoting breastfeeding at a migrant health center. Am J Public Health 1988; 78:523–525.
6. Smith JC, Mhango CG, Warren CW, *et al:* Trends in the incidence of breastfeeding for Hispanics of Mexican origin and the Anglos on the US-Mexico border. Am J Public Health 1982; 72:59–61.
7. Rassin DK, Richardson CJ, Baranowski T, *et al:* Incidence of breastfeeding in a low socioeconomic group of mothers in the US: Ethnic patterns. Pediatrics 1984; 73:132–137.
8. Forman MR: Review of research factors associated with choice and duration of breastfeeding in less developed countries. Task force on infant feeding practices. Pediatrics 1984; 74:667–694.

Pertussis Immunization in Eight-Month-Old Children in North Carolina

Dennis A. Clements, MD, MPH, Catherine M. Wilfert, MD, J. Newton MacCormack, MD, MPH,
Kristen A. Weigle, MD, MPH, and Floyd W. Denny, MD

Abstract: Between 1984 and 1987 reported pertussis cases in North Carolina increased threefold. Pertussis immunization rates were examined for those years in three one-year cohorts drawn from a random selection of North Carolina birth records. The percentage of children immunized with three DTPs at eight months of age was 58.1, 58.6, and 56.7 for the three cohorts. Only 20.5 percent of 117 reported pertussis cases in children 9–36 months of age during the last 10 years were adequately immunized. The low pertussis immunization rate may have contributed to the recent increase in pertussis cases in North Carolina. (*Am J Public Health* 1990; 80:734–735.)

Introduction

The United States has reported a recent increase in the number of pertussis cases.[1,2] In North Carolina the annual average of reported pertussis cases increased from 40 for the 10-year period 1975–85 to 88 and 123 cases in 1986 and 1987 respectively.

Although it is estimated that over 95 percent of North Carolina children entering primary school have the pertussis immunizations required recently by state law, a 1980 sample survey in North Carolina estimated that only 66 percent of two-year olds had received the recommended four DTP (diphtheria/tetanus/pertussis) injections.[3] Even less is known about the immunization status of children under one year of age who are at the greatest risk of severe pertussis disease and account for the majority of the cases reported and hospitalized.[4]

Our study was designed to examine the immunization status of eight-month old children in North Carolina during the time period 1984 to 1987, immediately preceding and during the pertussis epidemic.

Methods

A systematic random sample of 600 birth records was selected from all recorded North Carolina births between May 1, 1984 and April 30, 1985. Information on DTP immunization status was obtained using mailed questionnaires; with telephone or health department record follow-up for non-responders. From each of two birth cohorts: 5/1/85–4/30/86 and 5/1/86–4/30/87, 327 birth records were similarly selected. A 10 percent random sample was selected from those not responding for telephone interview. The telephone information was extrapolated to represent a similar fraction of the entire nonresponder group in each cohort to give an adjusted immunization rate.[5]

A child was considered properly immunized if three DTP injections were received by 244 days (eight months) of life. A few extra weeks beyond the recommended age (six months of age) for the third DTP immunization was allowed to avoid depressing the primary series completion rate by those who were only minimally delayed in obtaining immunization.

Confidence intervals for proportions were calculated using the normal approximation to the binomial distribution when n was sufficiently large. Hypothesis tests were performed using the normal approximation to the binomial for proportions, chi-square test statistic, Fisher's exact test or chi-square test for trend as appropriate.[6,7]

Results

The response rate in the 1985/86 and 1986/87 cohorts (41 percent and 49 percent, respectively) were lower than the response rate in the 1984–85 cohort (70 percent). Data were obtained by telephone from 44.4 percent of the 1985/86 and from 61.9 percent of the 1986/87 subsamples of nonresponders and used to adjust the final rates.

The adjusted immunization rates were similar for the three study years; 58.1 percent (1984/85), 58.6 percent (1985/86), and 56.7 percent (1986/87) (chi-square test for linear

---

Address reprint requests to Floyd W. Denny, MD, University of North Carolina School of Medicine, Program for HP/DP, CB# 7420, Chapel Hill, NC 27599. Dr. Clements and Dr. Weigle are affiliated with the Department of Epidemiology, UNC School of Public Health; Dr. Clements and Dr. Wilfert are affiliated with The Department of Pediatrics, Infectious Disease Division, Duke University Medical Center; Dr. MacCormack is with the North Carolina Department of Environment, Health and Natural Resources. This paper, submitted to the Journal March 28, 1989, was revised and accepted for publication October 19, 1989.

trend p = .737). Racial characteristics of the adjusted cohorts were similar to those of North Carolina in 1985 and did not play a role in the differences between cohort groups. There were significant differences, however, between the immunization rates for White (1984/85 63.2 percent, 1985/87 62.7 percent) and Black (1984/85 48.3 percent, 1985/87 39.9 percent) children in the adjusted rates.

There were 505 cases of pertussis reported in North Carolina between 1978–87, 350 (69.3 percent) were children less than one year of age (Table 1), the majority occurring in children less than nine months of age (Figure 1). Table 1 shows the immunization rates for the cases by age; 20.5 percent of children aged 9–35 months of age between 1978–87 and 21.4 percent of children for the years 1986/87 were adequately immunized. The median age of cases from 1978 to 1986 was five months and the median age for cases in 1986/87 was seven months.

Extrapolation from the World Health Organization (WHO) nomogram[8] for percent of cases vaccinated and vaccine efficacy (estimated to be 80 percent) gives a population vaccination rate of 55–60 percent.

Discussion

The DTP immunization rates for young North Carolina children as determined in this study were disturbingly low between 1984 and 1987, but remained constant over the three-year period. Therefore, our findings do not substantiate a decreasing rate of pertussis immunization during the study period. Due to the poor response rate, however, particularly in the 1985/87 cohorts, an undetected immunization rate change may exist. The finding that nonresponders located were less well immunized than the initial responders suggests that the rate of pertussis immunization may have been closer to 50 percent if all nonresponders had been contacted.

The recent increase in reported pertussis cases may be due to causes other than failure to immunize. Increased case reporting alone is unlikely because the number of children under one year of age with pertussis requiring admission to major hospitals in North Carolina also increased during 1986/87. The fact that over 40 percent of the reported cases of pertussis were under five months of age also suggests that the low immunization rate in young children was not the sole cause of the increasing incidence of pertussis. However, inadequate immunization of preschool-aged siblings could increase the chance of transmission to young infants. Immunizations are required to be up-to-date when children enroll at certified day care centers in North Carolina but continued updating of records is not normally required and may not be performed. A gradually cumulative increase in this pool of preschool-aged susceptibles could have propagated transmission to younger children during this epidemic.

TABLE 1—Immunization Status of Reported Pertussis Cases in North Carolina 1978–87

Age at Disease (months)	# DTPs Required	# Immunized	%
<2	0	NA/48	NA
2–4	1	53/161	32.9
5–8	2	27/113	23.9
9–21	3	18/84	21.4
22–36	4	6/33	18.2
37–72	4	11/16	68.8
>72	5	21/50	43.8
TOTAL		136/505	26.9

NA = not applicable. If the 48 children in this group are removed from the denominator because they probably would not have been immunized, the overall immunization rate in the total group would be 136/457, 29.8 percent.

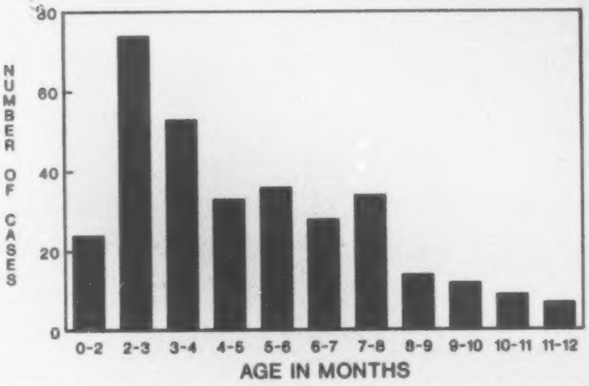


FIGURE 1—North Carolina Pertussis Cases Less than One Year of Age, 1978–87

Bass[1] has suggested that part of the reason for the recent increase in reported pertussis cases in the United States is that there is an increasing proportion of the population that is vaccine-immune to pertussis which is gradually losing its immunity. If older children and adults acquire asymptomatic infection, they could be a source of infection for others not yet immune, particularly unimmunized young children. It is of interest that the median age of pertussis cases increased from five months to seven months after 1985, coincident with the increase in the number of pertussis cases. It is also possible that an increasing use of day care facilities provided increased opportunities for incompletely immune children to be exposed to asymptomatically infected children and adults.

The increase in the number of pertussis cases and the delay in immunization that this and other studies[9] document is cause for concern. The disparity between the immunization rate of White and Black children is also of concern. It is likely, however, that race is a surrogate for socioeconomic status.

The task now is to better define groups of children that are poorly immunized, evaluate the barriers that prevent timely immunization, and formulate appropriate interventions.

ACKNOWLEDGMENTS

This study was funded in part by the Duke Perry Como Children's Fund and Dr. George Uhde. The data were presented at the Australasian Society of Infectious Diseases meeting March 23, 1989.

REFERENCES

1. Bass JW, Stephenson SR: The Return of Pertussis. Pediatr Infect Dis J 1987; 6:141–144.
2. US Department of Health and Human Services, Public Health Service, Epidemiology Program Office, Centers for Disease Control, Atlanta, Georgia: Summary of Notifiable Diseases United States 1987; 36(54):55.
3. NC Department of Human Resources, Division of Health Services: North Carolina Immunization Survey of Two-Year-Old Children, April 1980.
4. MMWR Reports, Pertussis Surveillance, United States 1984 and 1985. JAMA 1987; 257:2013–2014.
5. Cochran WG (ed): Sampling Technique, 3rd Ed. New York: John Wiley and Sons, 1976; 141, 359–411.
6. Remington RD, Schork MA (eds): Statistics with Applications to the Biological Sciences. Englewood Cliffs, NJ: Prentice-Hall, 1985.

7. Schlesselman JJ (ed): Case-Control Studies, Design, Conduct, Analysis. New York: Oxford University Press, 1982.
8. Orenstein WA, Bernier RH, Hinman AR: Assessing vaccine efficacy in the field, further observations. Epidemiol Rev 1988; 10:212–241.
9. Lewis T, Osborn LM, Lewis K, Brockert J, Jacobsen J, Cherry JD: Influence of Parental Knowledge and Opinions on 12-Month Diphtheria, Tetanus and Pertussis Vaccination Rates: Am J Dis Child 1988; 142:283–286.

Loss of Maternally Acquired Measles Antibodies in Well-Nourished Infants and Response to Measles Vaccination, Peru

ABRAHAM VAISBERG, PHD, JOSE O. ALVAREZ, PHD, HERMINIO HERNANDEZ, MD, DANIEL GUILLEN, MD, PABLO CHU, BS, AND ANA COLAROSSI, MS

Abstract: Clinical, anthropometric, and serological evaluations were conducted at birth and at 3, 6, 9, and 10 months (post measles vaccination) in 34 well-nourished Peruvian infants. Seroconversion rate after measles vaccination was 94 percent. The rate of antibody loss was a direct function of birth titer; at age 9 months, all children had identical mean titers regardless of their titer at birth. Differences in maternally acquired measles antibodies at birth were important only during the first six months of life. (*Am J Public Health* 1990; 80:736–738.)

Introduction

Infants in developing countries become susceptible to measles infection at younger ages than do infants in the industrialized nations.[1–3] Seroconversion rates after measles vaccination among infants younger than 9 months of age in Africa and Latin America are higher than in US infants of the same age.[4,5] Thus, maternally acquired antibodies against measles do not appear to confer the same length of protection in third world infants as in infants from developed countries.

It has been proposed that a lower maternal level of antibody, hence a lower circulating level in the newborn, may be responsible for the early susceptibility to measles infection in infants from low-income regions.[6] Alternatively, Halsey, *et al*,[7] have suggested that third world infants may have an increased rate of measles antibody loss. Elucidation of the reasons for early susceptibility to measles may allow design of more appropriate vaccination schedules.

Methods

The investigation was conducted in San Martin de Porres, a poor district of the city of Lima, Peru. One hundred full term, normal birthweight infants born at Cayetano Heredia Hospital were included in this longitudinal study at the time of birth. The parents agreed to participate in this investigation and brought their children to the hospital for evaluations every three months. Children were examined by a physician at birth and at 3, 6, 9, and 10 months of age. At every visit, mothers were interviewed to assess past infection with measles and other major illnesses. At age 9 months, all participating children were vaccinated against measles and one month later their response to the vaccine was assessed.

Weight and height measurements were made at each visit. Weight for age, weight for length, and length for age were calculated using the National Center for Health Statistics and Waterlow's classification.[8,9]

A venous blood sample was obtained from the mothers at the time of delivery. A small blood sample was obtained from each child at the time of recruitment and at each visit. The last sample (at 10 months) was used to assess seroconversion to measles vaccination. All samples were frozen at −70 °C until processed together.

Circulating IgG specific against measles was measured by ELISA using measles-infected Vero cells and peroxidase-conjugated rabbit immunoglobulin to human IgG according to the method originally described by Rice, Casali and Oldstone.[10] Each serum was diluted serially in saline containing 10 percent fetal bovine serum and 0.05 percent Tween 20 and aliquots of each dilution were assayed. Absorbance was read using a manual EIA plate reader. Positive and negative control sera were included in each assay plate. Also, negative control wells using uninfected Vero cells were included in all assay plates. Absorbance was plotted against log of serum dilution. Using linear regression, the intercept with the abcisa was determined and this dilution value, at zero absorbance, was taken to be the titer.

Attenuated live measles virus vaccine (Connaught, manufactured in Canada) was obtained from the Ministry of Health of Peru and stored at 4C. Each dose contained not less than 1,000 TCID50 of measles virus.

Results

Thirty-four subjects, 17 boys and 17 girls, completed the study and only the samples corresponding to these infants were processed. The nutritional status of the children was normal throughout the study with the exception of three girls who showed mild wasting at 3 months of age but recuperated quickly.

Table 1 shows the mean circulating measles antibody titers for mothers and infants at birth and at 3, 6, and 9 months of age and at post vaccination (10 months of age).

In order to follow the decline of maternally acquired measles antibody, infants were grouped into high, intermediate, and low birth titer. Mean levels of measles antibody were plotted as a function of age for the three groups of infants as shown in Figure 1. Infants with higher birth titers

---

Address reprint requests to Jose O. Alvarez, PhD, Professor and Director, Division of International Health and Public Health Nutrition, Departments of Public Health and Nutrition Sciences, School of Public Health, 303 TH, University of Alabama, Birmingham, AL 35294. All co-authors are with the Departments of Microbiology, Pediatrics, and Biochemistry, Universidad Peruana Cayetano Heredia, Lima, Peru. This paper, submitted to the Journal December 12, 1988, was revised and accepted for publication August 30, 1989.

TABLE 1—Mean Geometric Measles Antibody Titers (GMT)[†*]

	Boys	Girls	All
Mother	1502 ± 211 (7.31)	1622 ± 374 (7.39)	1561 ± 217 (7.35)
Infants			
at birth	2538 ± 400 (7.84)	1833 ± 580 (7.51)	2146 ± 357 (7.67)
at 3 months	393 ± 37 (5.97)	341 ± 70 (5.83)	364 ± 41 (5.90)
at 6 months	244 ± 40 (5.50)	228 ± 27 (5.43)	236 ± 24 (5.46)
at 9 months	80 ± 12 (4.38)	72 ± 8 (4.28)	76 ± 7 (4.33)
Post-vaccination	3290 ± 484 (8.10)	2975 ± 515 (8.00)	2982 ± 370 (8.00)

[†] mean geometric titer ± standard deviation
[*] values in parentheses = log2 GMT

TABLE 2—Half-life of Maternally Acquired Measles Antibody for Infants grouped into Low, Intermediate, and High Birth Titer

Group	n	Birth Titer [†]	Half-life (days)[*]	Post-Vaccination Titer
1	15	1000–2000 (1387 ± 64)	61 ± 13	3588 ± 489
2	9	2000–3000 (2346 ± 88)	59 ± 15	4098 ± 781
3	10	>3000 (6289 ± 1021)	46 ± 16	4067 ± 1404

[*] Calculated using circulating antibody titers at 3 months of age.
[†] Number in parentheses is mean of geometric titer at birth ± standard error for each group.

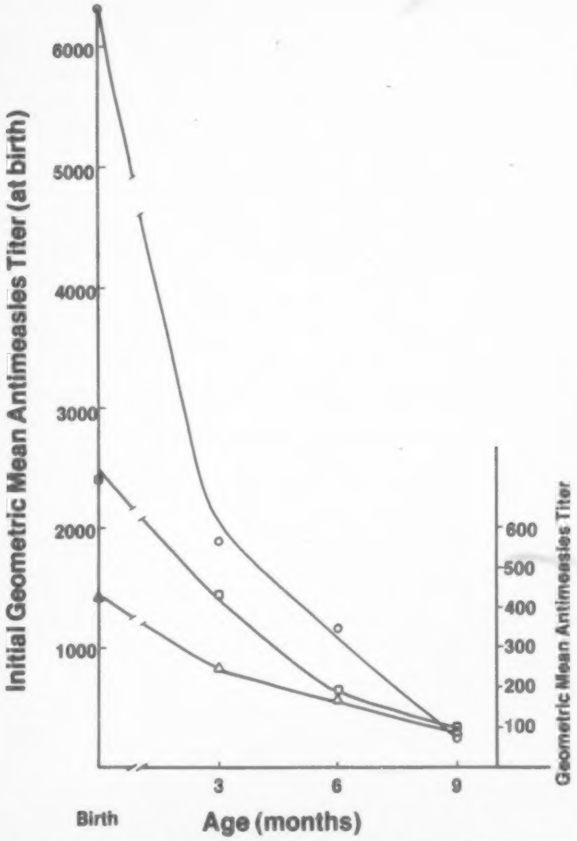


FIGURE 1—Geometric Mean Antimeasles Titer at Birth (left ordinate) and at 3, 6 and 9 Months (right ordinate) Plotted as a Function of Age
The break in the lines indicates that the points to the right of the break correspond to the scale on the right ordinate. Open circles (○) correspond to the group of infants with a high birth titer; open squares (□) to infants with intermediate birth titer; open triangles (△) to infants with low birth titer.

lost their antibodies more rapidly than infants with lower birth titers.

The half-life of maternally acquired measles antibody, calculated as described by Lennon and Black,[11] is shown in Table 2. The mean half-life for the three groups of infants was inversely related to birth antibody titer.

Seroconversion rate after measles vaccination was 94 percent among these children. Due to the method used to calculate antibody titer that led to a high sensitivity for our ELISA assay, seroconversion was arbitrarily defined as a post-vaccination titer above 1,000 and at least an order of magnitude higher than the prevaccination value.

Discussion

Our results show that during the first three to six months of life, infants born with higher levels of measles antibody are better protected against the disease than infants born with lower titers. After 6 months of age this difference becomes tenuous and disappears completely by age 9 months.

Our results are in agreement with several studies that have suggested that the rate of degradation of circulating antibodies depends on the total IgG concentration.[12,13] This phenomenon has been reported in mice,[14,15] rabbits,[16] and human beings.[17–19] The half-life of passively administered gammaglobulin in children with agammaglobulinemia was 50–60 days whereas, in normal children, it was 20–40 days.[20] Cloonan, Hawkes and Stevens[21] have reported that babies having high rubella titers at birth lose their antibody rapidly, whereas in babies with low birth titers, antibody declines over a longer period of time.

The antibody half-life estimate from our data, 56.3 days, is similar to that calculated by Black, *et al*,[6] for Taiwanese children (53.3 days) but longer than that observed in children from Kenya (46.8 days).[22]

It is interesting to note that boys showed higher antibody titers than girls at all ages, although this may not hold up with larger samples.

Black, *et al*.[6] have used maternal and cord measles data from 15 populations from nine countries to demonstrate a strong inverse correlation between per capita income and the age at which children respond to measles vaccination. The authors have observed also a correlation between maternal antibody titer and the age at which children seroconverted. These findings led them to postulate that children in developing countries become susceptible to measles infection at an earlier age than children in industrialized countries because they receive less antibodies from their mothers.

Our study, even though it involved a small number of healthy and well-nourished children, suggests that infants appear to have equal protection after eight months of age regardless of what their antibody level was at birth. The fact that no correlation was observed between infant titer at birth and post vaccination titer strengthens the validity of our findings. If the length of protection conferred by maternal antibody as suggested by Black, *et al*,[6] were primarily a consequence of infant titer at birth, one would expect vaccination titer to be an inverse function of birth titer. This was not the case in our study. Other factors are probably involved in the early loss of passive measles immunity in children from developing countries.

ACKNOWLEDGMENTS

The financial support of the Consejo Nacional de Ciencia y Tecnologia (CONCYTEC) of Peru and the Instituto Hipolito Unanue are gratefully acknowledged.

REFERENCES

1. Morley DC: Severe measles in the Tropics. Br Med J 1969; 1:363–365.
2. O'Donovan C: Measles in Kenyan children. East Afr Med J 1971; 48:526–532.
3. Pan American Health Organization: Proceedings on application of vaccines against viral, rikettsial and bacterial diseases of man. Sci Publ 1971; 226:209.
4. Halsey NA: The optimal age for administering measles vaccine in developing countries. *In:* Halsey NA, de Quadros CA, (eds): Recent Advances in Immunization. Washington, DC: Pan American Health Organization, 1983.
5. Black FL: The role of herd immunity in control of measles. Yale J Biol Med 1982; 55:351–360.
6. Black FL, Berman LL, Borgono, JM, Capper RA, Carvalho AA, Collins C, Glover O, Hijazi Z, Jacobson DL, Lee YL, Libel M, Linhares AC, Mendizabal-Morris CA, Simoes E, Siqueira-Campos E, Stevenson J, Vecchi N: Geographic variation in infant loss of maternal measles antibody and in prevalence of rubella antibody. Am J Epidemiol 1986; 124:442–452.
7. Halsey NA, Boulos R, Mode F, Andre J, Bowman L, Yaeger RG, Toureau S, Rohde J, Boulos C. Response to measles vaccine in Haitian infants 6 to 12 months old. N Engl J Med 1985; 313:544–549.
8. Waterlow JC: Classification and definition of protein-calorie malnutrition. Br Med 1972; 3:566–569.
9. Waterlow JC, Buzina R, Keller W, Lane JM, Nichaman MZ, Tanner JM: The presentation and use of height and weight data for comparing the nutritional status of groups of children under the age of 10 years. Bull WHO 1977; 55:489–498.
10. Rice GPA, Casali P, Oldstone MBA: A new solid phase enzyme linked immunosorbent assay for specific antibodies to measles virus. J Infect Dis 1983; 147:1055–1059.
11. Lennon VL, Black FL: Maternally derived measles immunity in sera of vaccine-protected mothers. J Pediatr 1986; 108:671–676.
12. Brambell FWR: The transmission of immunity from mother to young and the catabolism of immunoglobulins. Lancet 1966; 2:1087.
13. Brambell FWR, Hemmings WA, Morris IG: A theoretical model of gammaglobulin catabolism. Nature 1964; 203:1352–1355.
14. Fahey JL, Robinson AG: Factors controlling serum gamma-globulin concentration. J Exp Med 1963; 118:845–868.
15. Fahey JL, Sell S: The immunoglobulin of mice. V. The catabolic properties of 5 immunoglobulin classes. J Exp Med 1965; 122:41–58.
16. Anderson SB, Bjornebre M: Gammaglobulin turnover in rabbits before and during hyperimmunization. J Exp Med 1964; 119:537–546.
17. Solomon A, Waldmann TA, Fahey JL: Metabolism of normal 6.6S gammaglobulin in normal subjects and in patients with macroglobulinemia and multiple myeloma. J Lab Clin Med 1963; 62:1–17.
18. Waldmann TA, Schwab PJ: IgG metabolism in hypogamma-globulinemia: Studies in patients with defective gammaglobulin synthesis. J Clin Invest 1965; 44:1523–1533.
19. Waldmann TA: Disorders of immunoglobulin metabolism. N Engl J Med 1969; 281:1170–1177.
20. Gitlin D, Janeway JA, Apt L, Craig JM: Agammaglobulinemia. *In:* Lawrence HS (ed): Cellular and Humoral Aspects of the Hypersensitive State. New York: Hoeber-Harper, 1959; 375.
21. Cloonan MJ, Hawkes RA, Stevens LH: Postnatal decline of maternally acquired rubella antibodies. J Hyg Camb 1970; 68:461–468.
22. Ministry of Health of Kenya, World Health Organization: Measles immunity in the first year after birth and the optimum age for vaccination in Kenyan children. Bull WHO 1977; 55:21–31.

27

The Prime Time Diet: A Content Analysis of Eating Behavior and Food Messages in Television Program Content and Commercials

Mary Story, PhD, RD, and Patricia Faulkner, MS

Abstract: The purpose of this study was to identify and analyze messages related to food and eating behavior as presented on prime time television (8:00–11:00 pm) both in programming and commercials. Food references occurred an average of 4.8 times per 30 minutes of programming time. Over half (60 percent) of all food references in programs were for low nutrient beverages and sweets. The prime time diet is inconsistent with dietary guidelines for healthy Americans. (*Am J Public Health* 1990; 80:738–740.)

Introduction

The American population spends more time watching television than it spends in any other activity except sleep and work.[1] There is increasing concern about the impact of this powerful and pervasive medium on health and health-related behaviors.[1–6] While the exact nature of the impact of television on behavior is controversial and largely unstudied, there is no doubt that many health-related images and messages are constantly conveyed to viewers, and may influence health behaviors. Because of the accumulating knowledge of the relationship of diet to health and chronic diseases, we thought it would be interesting to see how food and eating behaviors are portrayed on television, and whether these messages and behaviors are consistent with dietary recommendations for good health. The purpose of our study was to identify television messages related to food and eating behaviors both in programs and in commercials. Prime time television (8:00–11:00 pm) was selected for content analysis because it attracts the largest viewing audience among all age groups, with the exception of preschoolers.

Methods

The 1988 Nielsen Report on Television[7] was used to select the top nationally ranked prime time evening network programs for analysis. From the 15 top ranked shows among total US television households, only those featuring dramatic or situational comedy series were selected for analysis. Eleven programs were analyzed: the Bill Cosby Show; A Different World; Cheers; Growing Pains; Who's The Boss; Murder She Wrote; Golden Girls; L.A. Law; Moonlighting; Family Ties; and Alf.

Each program was video taped on two separate occasions during a nine-week period in the summer of 1988. A trained assistant viewed each video tape and coded all references to food and all eating occasions in programs and

---

From the Division of Human Development and Nutrition and the Adolescent Health Program, University of Minnesota School of Public Health. Address reprint requests to Mary Story, PhD, RD, Assistant Professor, Division of Human Development and Nutrition, School of Public Health, University of Minnesota, Box 197 Mayo Memorial Building, 420 Deleware Street, SE, Minneapolis, MN 55455. This paper, submitted to the Journal August 4, 1989, was revised and accepted for publication November 30, 1989.

---

commercials on an instrument adapted from one developed by Kaufman.[8] The coding was checked for reliability by double coding a subsample of programs by an independent observer. For each reference to food or drink in programming, the coder identified the food or drink involved, whether it was a verbal or visual reference, or an actual eating scene, and the context in which food was eaten (meal or snack). Incidental visual references to food (e.g., a basket of fresh fruit on a kitchen counter) were counted as food references. Verbal references using food in a figurative sense, such as referring to someone as "the big cheese" were not included. All commercials aired during the show were recorded but only those advertising food or beverages were analyzed. The following information was coded: the product name and company, any explicitly expressed messages or claims about the food being advertised, and food shown other than that being advertised.

Results

Food references in prime time shows (excluding commercials) occurred an average of 4.8 times per 30 minutes of programming time (134 food references). References to low nutrient beverages (coffee, alcohol, soft drinks) occurred at a higher rate than any other category of food (Table 1). Over half (60 percent) of all food references in prime time programs were for low nutrient beverages and sweets.

TABLE 1—Rates of Occurrences of References to Food in Television Programs by Food Category*

Food Category	Total References (n)	Rate (per Half Hour)
Low-Nutrient Beverages		
Coffee	26	0.93
Alcohol	18	0.64
Soft drinks	12	0.43
Total	56	2
Sweets		
Cakes, cookies, desserts	13	0.46
Donuts, sweet rolls	8	0.28
Candy	2	0.07
Ice cream	1	0.04
Total	24	0.86
Meats		
Beef	9	0.32
Poultry	1	0.04
Fish	1	0.04
Sandwiches	6	0.21
Total	17	0.61
Fruits and Vegetables		
Fruits	3	0.11
Fruit juice	3	0.11
Vegetables	7	0.25
Total	13	0.46
Salty Snacks		
Popcorn	4	0.14
Peanuts	3	0.11
Pretzels	2	0.07
Total	9	0.32
Breads and Cereals		
Cereal	4	0.14
Breads	2	0.07
Muffins	1	0.04
Crackers	1	0.04
Total	8	0.29
Dairy Products		
Cheese	2	0.07
Milk	5	0.18
Total	7	0.25

*A total of 28 half-hour segments were viewed.

TABLE 2—Number and Rates of Food Commercials Shown during Prime Time Television Programs by Category*

Types of Food Advertised	Total References (n)	Rate per 30 minutes	Rate per Minute
Restaurants			
Fast food	23	0.82	0.10
Family style	1	0.04	0.00
Low-Nutrient Beverages			
Soft drinks, regular	7	0.25	0.03
Soft drinks, diet	4	0.14	0.02
Coffee/tea	3	0.11	0.01
Beer/wine	5	0.18	0.02
Cereals			
Sugared	1	0.04	0.00
Unsugared	17	0.61	0.06
Sweets/Desserts			
Ice cream, frozen treats	5	0.18	0.02
Cake, candy, cookie, etc.	7	0.25	0.03
Entrees			
Convenience entrees	5	0.18	0.02
Dairy Products			
Cheese	1	0.04	0.00
Yogurt	3	0.11	0.01
Fruits			
Orange juice	2	0.07	0.01
Raisins	1	0.04	0.00
Salty Snacks	2	0.07	0.01
Miscellaneous			
Butter	2	0.07	0.01
Condiments	2	0.07	0.01

*A total of 28 half-hour segments were viewed.

In the 22 shows, there were 86 occasions of eating or drinking. Almost three-fourths (72 percent) of the food eaten on prime time shows is eaten between meals. Snacking occurred at a rate of 2.2 times per half hour of programming versus .86 times for meals. The most frequent snack food was sweets (44 percent), followed by salty snack foods (25 percent). Less than 10 percent of snacks were fruits (3 percent) or vegetables (6 percent). Coffee (43 percent) was the most frequently consumed beverage, followed by alcohol (28 percent) and soft drinks (17 percent).

Of the 24 episodes involving meals, 12 were evening meals and six were breakfast and six lunch. Breakfast foods consisted of either cereal and toast (50 percent) or sweet rolls and donuts (50 percent). Fruit was shown three times at breakfast and once at lunch. In almost one-third (33 percent) of the noon and evening meal episodes, the only food portrayed was dessert (with either coffee or wine). Other foods shown for these meals were sandwiches (28 percent); salads (11 percent); entrees (28 percent), consisting of stir fry vegetables and chicken which the children refused to eat; lobster with butter; hot dogs and veal with potatoes. One-third of all meal episodes included alcohol.

Of the 261 commercials aired, 91 (35 percent) were food ads. Table 2 shows the number and rate of food references in commercials by food category. Commercials for fast food restaurants occurred at a higher rate than any other food category. The fast foods advertised were hamburgers, fried chicken, pizza, and breakfast sandwiches. None of these commercials advertised salads or salad bars. Only three commercials advertised fruit, and none advertised vegetables; however, fruits and vegetables were incidentally shown in 36 percent of all food ads (e.g., orange juice in an ad for breakfast cereals; fruit shown in an ad for candy).

The most frequently expressed message in commercials were claims of "good taste" and food being "fresh and natural." Few commercials made explicit nutritional claims. Those that did tended to be ads for unsweetened breakfast cereals, claiming that the products were nutritious, low in fat and sodium, and high in fiber.

Discussion

References to food were pervasive, both in prime time programs and in accompanying commercials. The "prime time diet" primarily consisted of foods low in nutritional quality, such as low nutrient beverages, sweets, and salty snack foods. Foods were typically consumed as snacks rather than meals. This reflects the eating pattern of the American population: diets high in sugar, sodium, and fat and low in fruits, vegetables, and fiber.[9–12] Recent data,[13,14] also indicate that both adults and children are snacking more frequently, and that families are eating fewer meals together. In light of the scientific evidence on the relationship of dietary factors to health and chronic disease risk, several studies, as well as numerous national reports and papers, have pointed to the wide-spread need for public education and changes in dietary behavior.[9–12,15]

Given our findings that messages related to eating practices are conveyed frequently during prime time television viewing, and that the television diet exemplifies poor nutritional practices, an important but unanswered question is whether television exposure to food references has any impact on actual eating behavior. Modeling and social learning theory would suggest that frequent, regular TV exposure might influence eating habits. However, this is speculative and further research is needed.

We believe media can be important in creating social norms and promoting healthy eating practices. Much of the eating on TV programs is trivial and incidental, used for "stage business," that is something to do with one's hands. Although producers, writers, and directors are in the business of delivering entertainment, and not with projecting health images, there is an opportunity to present health promoting messages by having characters model good dietary practices. Health professionals should initiate and work with the TV industry to encourage the inclusion of healthy eating patterns in programming.

ACKNOWLEDGMENTS

The authors thank Anne Ganey for technical assistance. This study was supported, in part, by the MCH Grant #MCJ-009118-01-0.

REFERENCES

1. Gerbner G, Larry G, Morgan M, Signorielli N: Health and medicine on television. N Engl J Med 1981: 305:901–904.
2. Tucker LA, Friedman GM: Television viewing and obesity in adult males. Am J Public Health 1989, 79:516–518.
3. Singer DG: Children, adolescents and television—1989: 1. Television violence: A critique. Pediatrics 1989; 83:445–446.
4. Breed W, De Foe JR: Drinking and smoking on television 1950–1982. J Public Health Policy 1984; 5:257–270.
5. Wallack L, Breed W, Cruz J: Alcohol on Prime-Time Television. J Stud Alcohol 1987; 48:33–38.
6. Hansen A: The portrayal of alcohol on television. Health Educ J 1986; 45:127–131.
7. Nielsen Media Research: 1988 Report on Television. Northbrook, Illinois: AC Nielsen Co, 1988; 1–17.
8. Kaufman L: Prime time nutrition. J Commun 1980; 30:37–46.
9. Block G, Rosenberger W, Patterson B: Calories, fat and cholesterol: Intake patterns in the US population by race, sex and age. Am J Public Health 1988; 78:1150–1155.
10. Patterson BH, Block G: Food choices and the cancer guidelines. Am J Public Health 1988; 78:282–286.
11. US Department of Health and Human Services, Public Health Service: The Surgeon General's Report on Nutrition and Health. DHHS Pub. No. (PHS) 88-50210. Washington, DC: Govt Printing Office 1988; 712.
12. Committee on Diet and Health, Food and Nutrition Board, National Research Council: Diet and Health: Implications for Reducing Chronic Disease Risk. Washington, DC: National Academy Press 1989; 1–749.
13. Nationwide Food Consumption Survey. Continuing Survey of Food Intakes by Individuals. Women 19–50 Years and Their Children 1–5 Years, 1 Day NFCS, CSFII, Report No. 85-1. Washington, DC: US Department of Agriculture, 1985.
14. Nationwide Food Consumption Survey. Continuing Survey of Food Intakes by Individuals. Men 19–50 Years, 1 Day. NFCS, CSFII Report No. 85-3. US Department of Agriculture, 1985.
15. Crawford P: The nutrition connection: Why doesn't the public know? (editorial) Am J Public Health 78:1147–1148.

NIH Consensus Panel Issues Conference Report on Treatment of Destructive Behaviors in Persons with Developmental Disabilities

In September 1989, the National Institute of Child Health and Human Development co-sponsored a Consensus Development Conference on "Treatment of Destructive Behaviors in Persons with Developmental Disabilities." The panel's report, which contains recommendations and conclusions concerning treatment of such behaviors, has been printed and single copies are available free from: William H. Hall, Director of Communications, Office of Medical Applications of Research, National Institutes of Health, Building 1, Room 259, Bethesda, MD 20892. Tel: 301/496-1143.

27

Seat Belt Use on Interstate Highways

JoAnn K. Wells, BA, Allan F. Williams, PhD, Adrian K. Lund, PhD

Abstract: More than 5,000 miles of limited-access highways in the eastern United States and Canada were traveled to observe seat belt use. Overall belt use was 58 percent in the United States and 79 percent in Canada. The data indicate that belt use in the United States follows a different pattern on interstate highways than on other streets and roads, with relatively high belt use rates (over 50 percent) appearing to be somewhat independent of belt use law provisions. (*Am J Public Health* 1990; 80:740–742.)

Introduction

Information on seat belt use is typically based on observations of motorists on city streets. For example, the National Highway Traffic Safety Administration (NHTSA) has surveyed belt use in 19 major cities annually since 1975.[1] However, belt use is known to vary according to where observations are made, and one location where belt use typically is higher than on city streets is interstate highways. In surveys in four states, belt use was 10–20 percentage points higher on interstate highways connecting the cities than in the cities.[2] Systematic information on seat belt use in vehicles traveling outside cities is lacking. In the four-state study it was collected incidentally as observation teams traveled between cities.[2] The present study provides information on belt use on interstate highways in the eastern United States and compares use rates in states and Canadian provinces with and without belt use laws.

Methods

Seat belt use observations were made by trained observers sitting behind the driver of a passenger van that traveled slower than the prevailing traffic in the far right lane. From that location, observers could clearly see shoulder belt use of front seat occupants of cars, light trucks, and vans passing in the adjacent lane. Lap belt use was not observed. Most cars on the road (all vehicles beginning with model year 1974) have integral lap/shoulder belts in the driver and right front seating positions. However, pre-1974 cars were not excluded so belt use is slightly underestimated.

Belt use observations were made on 5,129 miles of interstate highways in 21 eastern states and on limited-access highways in two eastern Canadian provinces from 9 am to 4 pm weekdays in August 1988. Appendix I provides information on the jurisdictions and highways surveyed and belt use law status.

It should be noted that the kinds of travel on these highways can vary tremendously from state to state. For example, in New York state, I-95 is entirely urban with short distances between access points and large volumes of local traffic; in South Carolina the same interstate is entirely rural with relatively long distances between access points.

Observed differences in belt use for different populations were tested by χ^2 test of independence; only differences $\alpha = 0.05$ are reported.

Results

Seat belt use by the 14,354 front seat occupants observed on the highways surveyed averaged 58 percent in the United States and 79 percent in Canada. Belt use ranged from 48 percent to 66 percent in the 21 states and was 76 percent in Ontario and 84 percent in Quebec (Table 1). In the United States use was higher in states with laws (60 percent) than states without laws (56 percent), but the range of use rates for states with and without laws overlapped considerably. There was no significant difference in belt use between states with laws permitting primary and secondary enforcement.

The majority of occupants (57 percent) were in vehicles registered in the state or province in which they were observed. Of those traveling in out-of-state vehicles, motorists from jurisdictions with seat belt laws were more likely to use belts than motorists from non-law jurisdictions, whether observed in states with laws (66 vs 57 percent), provinces with laws (81 vs 74 percent), or non-law states (66 vs 56 percent).

Belt use was higher among drivers; in cars, utility vehicles, and passenger vans; among those estimated to be age 25 or older; and among women (Table 2).

Discussion

Belt use on interstates is higher than belt use in cities in the same state. Even in states without laws, belt use on interstates was greater than 50 percent, about 20 percentage

TABLE 1—Interstate Belt Use, by Jurisdiction

Jurisdiction	Belt Use %	 (N)	Standard Error of Percent Belt Use
United States			
Law	60	(7,943)	0.6
Connecticut	58	(526)	2.2
Florida	56	(2,149)	1.1
Illinois	55	(257)	3.1
Indiana	58	(311)	2.8
Maryland	66	(697)	1.8
Michigan	59	(581)	2.0
New Jersey	65	(477)	2.2
New York	54	(98)	5.0
North Carolina	59	(490)	2.2
Ohio	64	(453)	2.3
Pennsylvania	66	(605)	1.9
Tennessee	51	(476)	2.3
Virginia	63	(823)	1.7
No Law	56	(3,311)	0.9
Delaware	64	(59)	6.2
Georgia	59	(1,265)	1.4
Kentucky	64	(140)	4.1
Maine	52	(711)	1.9
Massachusetts	49	(404)	2.5
New Hampshire	54	(63)	6.3
Rhode Island	48	(212)	3.4
South Carolina	60	(457)	2.3
Canada			
Ontario	76	(2,042)	0.9
Quebec	84	(1,058)	1.1

From the Insurance Institute for Highway Safety. Address reprint requests to JoAnn K. Wells, Insurance Institute for Highway Safety, 1005 N. Glebe Road, Arlington, VA 22201. This paper, submitted to the Journal July 14, 1989, was revised and accepted for publication October 3, 1989.

TABLE 2—Interstate Seat Belt Use by Front Seat Occupants by Vehicle and Motorist Age, Sex, and Seating Position

Characteristics	Belt Use %	(N)
Vehicle Type		
Car	66	(11,447)
Utility	61	(471)
Pickup	36	(1,100)
Passenger Van*	73	(663)
Other Vans	42	(644)
Unknown		(29)
Estimated Age (years)		
<25	56	(2,412)
25 or older	64	(11,921)
Unknown		(21)
Sex		
Female	66	(5,694)
Male	61	(8,620)
Unknown		(40)
Seating Position		
Driver	64	(9,519)
Right front passenger	61	(4,835)

*The following passenger vehicles in this category are: Nissan Van, Toyota Van, Mazda MPV, Chevrolet Astro, GMC Safari, Ford Aerostar, Dodge Caravan, Dodge Grand Caravan, Plymouth Voyager, Plymouth Grand Voyager, and Mitsubishi Van. The majority of these vans are sold as passenger vehicles, but the current data include observations of cargo versions as well.

points higher than in cities in these same states surveyed by NHTSA.[1] The laws do make a difference, however, not only in elevating use rates within the states, but motorists with vehicles registered in states with laws are more likely than motorists registered in states without laws to use seat belts wherever they travel. Nevertheless, variations in belt use laws affected belt use less on interstates than such laws are known to do on other highways, i.e., city-based surveys have found belt use to be higher in states with primary enforcement laws.[1,3] This should not be seen as an indication that belt use law differences are unimportant but that there is a different, and higher, pattern of belt use on interstates.

The reasons for higher interstate belt use are not clear. The population of motorists on interstates may be different than on other roads; for example, on average they may be of higher socioeconomic status, which is strongly related to belt use.[4] Also, people often claim in surveys that they are more likely to buckle up for long trips on high speed highways, although no observational studies have adequately tested this claim.[5]

Use rates are high both in Canadian provinces in general and on their limited-access highways.[6] Belt use in Canada was less than 50 percent in the 1970s.[7] Extensive use of programs combining publicity and enforcement during the past decade is thought largely responsible for Canada's present high belt use.[8,9] Such programs have been run successfully on a limited basis in the United States but have not been used extensively enough to increase overall belt use.[10,11]

ACKNOWLEDGMENT

This work was supported by the Insurance Institute for Highway Safety.

APPENDIX I
Highways and Jurisdictions Surveyed

Jurisdictions	Routes	Mileage	Belt Use Law* at Time of Survey
United States			
Connecticut	I-95	119	Primary
Delaware	I-95, I-295	16	No
Florida	I-75, I-95	859	Secondary
Georgia	I-75, I-95	499	No
Illinois	I-94, I-57, I-24	371	Secondary
Indiana	I-94, I-80	44	Secondary
Kentucky	I-24	101	No
Maine	I-95	298	No
Maryland	I-495, I-270, I-70, I-95	225	Secondary
Massachusetts	I-95	91	No
Michigan	I-75, I-94	283	Secondary
New Hampshire	I-95	20	No
New Jersey	NJ Turnpike, I-95	136	Secondary
N. Carolina	I-95	180	Primary
New York	I-95	23	Primary
Ohio	I-76, I-80, I-80/90. I-75	224	Secondary
Pennsylvania	I-70, I-70/76, PA Turnpike, I-76	192	Secondary
Rhode Island	I-95	38	No
S. Carolina	I-95	193	No
Tennessee	I-24	199	Secondary
Virginia	I-495, I-95	192	Secondary
Canada			
Ontario	Rt. 401	512	Primary
Quebec	Rt. 20	314	Primary

*Primary: Motorist can be stopped or cited for a seat belt law violation alone. Secondary: Motorist can be cited for seat belt law violation only if stopped for other traffic offense.

REFERENCES

1. Bowman BL, Rounds DA: Restraint System Usage in the Traffic Population. DOT HS 807 342. Washington, DC: National Highway Traffic Safety Administration, 1987.
2. Williams AF, Wells JK, Lund AK: Shoulder belt use in four states with belt use laws. Accident Anal Prev 1987; 19:251–260.
3. Campbell BJ: 1985–1986 Experience with Belt Laws in the United States. Chapel Hill, NC: University of North Carolina Research Center, 1987.
4. Lund AK: Voluntary seat belt use among US drivers: Geographic, socioeconomic and demographic variation. Accident Anal Prev 1986; 18:43–50.
5. Mayas JMB, Boyd NK, Collins MA, Harris BI: A Study of Demographic, Situational and Motivational Factors Affecting Restraint Usage in Automobiles. DOT HS 806-402. Washington, DC: National Highway Traffic Safety Administration, 1983.
6. Transport Canada: Seat Belt Use Survey Leaflet (CL 8901 (E)). Ottawa, January 1989.
7. National Highway Traffic Safety Administration: Effectiveness of Safety Belt Use Laws: A Multinational Examination. DOT HS 8070181 Washington, DC: US Dept of Transportation, 1986.
8. Williams AF, Lund AK: Seat belt use laws and occupant crash protection in the United States. Am J Public Health 1986; 76:1438–1442.
9. Jonah BA, Dausin NE, Smith GA: Effects of a selective traffic enforcement program on seat belt usage. J Appl Psychol 1982; 67:89–96.
10. Williams AF, Lund AK, Preusser DF, Blumberg RD: Results of a seat belt use law enforcement and publicity campaign in Elmira, New York. Accident Anal Prev 1987; 19:243–249.
11. Lund AK, Stuster J, Flemming A: Special publicity and enforcement of California's belt use law: Making a "secondary" law work. J Criminal Justice 1989; 17(5):329–341.